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SOME PATHOLOGICAL CHANGES IN AUSTRALIAN ABORIGINAL BONES.

By CHARLES V. MACKAY, M.D. (Melbourne), F.R.A.C.P.,

Acting Director, Australian Institute of Anatomy, Canberra.

IN THE MEDICAL JOURNAL OF AUSTRALIA of May 30, 1936, there appeared an original article entitled "A Critical Survey of Some References to Syphilis and Yaws among the Australian Aborigines". This was written by Cecil J. Hackett. The bibliography contains fifty references, and is of a special character, showing evidence of considerable authoritative research.

Some of the conclusions were as follows:

It is suggested that, if *granuloma pudendi* is excluded, in most cases the disease referred to is yaws. In a future paper it will be pointed out that among the aborigines of the Northern Territory of Australia today syphilis is unknown, while yaws is frequent.

It will also be shown that in museums in Australia there are aboriginal bones from the southern and eastern areas which show yaws lesions. It is probably correct to conclude that when Australia was discovered by Europeans yaws was present in all native communities.

In the following account of my investigations into the pathology of aboriginal bones found near the River Murray in southern New South Wales, I am chiefly concerned with the statement that "in Australian museums are bones from the southern and eastern areas which show yaws lesions".

Hackett has attempted to substantiate this statement in a monograph published by the Royal Society of Tropical Medicine and Hygiene in September, 1936.

In the museums of this institute we have many examples of pathological changes in aboriginal bones. I have particularly investigated 351 speci-

mens, and hope at some future date to embody the detailed result of this work in a special monograph.

A preliminary survey is now submitted of some of the pathological features which have been observed. The total number of bones examined was 351, comprising 325 of the skeleton proper, and 26 skulls. No evidence is available as to whether the burial sites from which the bones were obtained antedated the period of European occupation of this country.

All the limb bones are well represented. The main pathological features are the fractures, the examples of osteoarthritis, and those bones exhibiting inflammatory changes which are either confined to the periosteum or have spread throughout the whole bone.

The number of bones exhibiting osteoarthritic changes is surprisingly large. The joints involved are almost entirely the elbow and the knee, with the former predominating. The severity of the changes observed on and around the articular surfaces composing these joints is very noticeable. Evidently erosion of the articular cartilages of the joint has occurred, followed by exposure of the bone, with in some instances eburnation of the bony surfaces in contact. Extensive osteophytic formation has occurred at the margins of the joints, causing bony irregularity of an unusual type and extent.

In a limited number of the bones, twelve in all, some degree of bowing deformity is to be seen. This is particularly well marked in two femora from an adolescent, in which striking changes are seen. There are only five examples of the so-called boomerang leg or pronounced forward curvature of the tibia.

Fractures of the Limb Bones.

In the 78 fractures under review there was no evidence of any of the common fractures of civilized life; no Colles's fracture, no Pott's fracture and no fracture of the neck of the femur was found. Only one example of fracture of the lower third of the humerus was observed.

In the forearm union was universally good in the 54 examples, comprising 69% of all the fractures. However, in the humerus (six examples) and in the femur (four examples) the results are, with one exception, wholly bad, union being obtained only with deformity and shortening.

In the tibia the result was decidedly bad in three out of the six specimens; but in the fibula, out of eight examples, there was only one definite case of malunion.

These results would seem to fit in with the observed habits of the Australian natives. A protective support to the forearm could be simply applied and would not be altered in position by the exigencies of a nomadic existence. The humerus, however, is a difficult bone to splint and to keep in proper alignment at any time; while the femur and the tibia would be subjected to many stresses of a prejudicial character during marches *et cetera*.

The fibula is already partially splinted by the tibia, and healing of this bone would not present any great difficulties. The gross failures occurred where good results are notoriously difficult to obtain, even under the best conditions, namely, the middle of the shaft of the humerus and of the femur.

Osteoarthritis.

An examination of the 58 examples of osteoarthritis gives the following information.

In the bones of the arm and forearm 42 examples of this pathological change are found in a total of 58 bones; the percentage is 72. These changes all involve the elbow joint, and no other joint of the upper limb is affected.

In the bones of the leg 16 examples of osteoarthritic change are found, all involving the knee joint alone.

There are thus 42 specimens of osteoarthritis of the elbow joint and 16 of the knee joint; these are the only joint surfaces showing evidence of this inflammatory process. In no other joint of the skeleton proper are these definite pathological changes seen, although in several skulls in the aboriginal section of the osteological museum of this institute similar evidences, usually localized to one side only, are found at the temporo-mandibular articulation.

In a search for the distinguishing characteristics which would assist classification of these specimens of joint changes, the following distinctions have been adopted between the two inflammatory conditions of osteoarthritis and rheumatoid arthritis. Much confusion has always existed as regards the differentiation of these two joint states, and it is fully recognized that all classifications are personal and somewhat arbitrary.

Differences between Osteoarthritis and Rheumatoid Arthritis.

1. The ultimate changes which are considered to be characteristic of osteoarthritis are the following: (*a*) the erosion of the articular cartilages of a joint, followed by exposure of the bone with eburnation of the bony surfaces in contact; (*b*) the formation at the margins of the joint of irregular outgrowths of bone called osteophytes; (*c*) the view that osteoarthritis is a very chronic process, which may be subsequent to injury of the joint, and is often confined to one joint.

In the aboriginal specimens examined the changes are restricted to two joints, the elbow and the knee. It is possible that these joints would be the ones most likely to suffer direct injury or excessive strain in tribal combats or in hunting.

2. It is suggested that the lesions of rheumatoid arthritis, on the contrary, are characterized by the following conditions: (*a*) there is no formation of osteophytic outgrowths; (*b*) the infection in rheumatoid arthritis appears to be more of an acute character and to partake more of the nature of a disease process than a response to injury; (*c*) the synovial membrane is more definitely the

seat of disease than the articular cartilages or the bones themselves.

Typical Osteoarthritic Changes observed in the Bones comprising the Articular Surfaces of the Knee and Elbow Joints.

The Knee Joint.—In the 12 specimens of the distal extremity of the femur are seen to a minor degree evidences of eburnation of a portion of the bony articular surface. The most characteristic feature, however, is the "heaping-up" of bone at the articular margins, especially on the anterior aspect. This osteophytic or new bone formation is apparently an excessive response to a long-continued stimulus. The edges are in many instances sharp and rugged. The most noticeable appearance associated with this osteophytic outgrowth of irregular flattened elevations is the formation by them of a definite rim to the bone.

Figure I.

S.F. 25, 26 and 27. Three examples of osteoarthritis of the head of the radius, with the striking osteophytic outgrowth giving a mushroom effect.

In the four specimens of the proximal extremity of the tibia with alterations at the articulating surface, minor changes of an osteoarthritic character are seen. The characteristic "heaping-up" of osteophytic outgrowth is particularly noticeable on the inner and outer margins of the condyles.

The Elbow Joint.—There are 16 examples of osteoarthritic changes in the distal articular extremity of the humerus. In all the specimens the same pathological changes are found, but varying in degree. Great osteophytic proliferation is seen at the distal end of the humerus, around the articulating surfaces entering into the formation of the elbow joint. This is sometimes accompanied by definite eburnation of the articulating bony surfaces. (See Figure II.)

The osteophytic heaping-up is so pronounced in some instances as almost to enclose the epicondyles, and thus to obliterate the normal anatomical relationships of the bony parts. In other specimens the olecranon fossa is closed by osteophytic new bony growth.

The Ulna.—Osteoarthritic changes are found in fifteen specimens of the proximal extremity of the ulna, comprising the articular portion of the elbow joint. Eburnation of the bony surfaces composing the joint, and heaping up of osteophytic deposits around the articular surfaces of the olecranon and coronoid processes have occurred. (See Figure XXIII.)

The Radius.—In eleven bones are seen the changes which have occurred in the proximal end of the radius, in the neighbourhood of the elbow joint.

Figure II.

S.F. 20:22. Showing osteoarthritis of the lower end of the left humerus on its anterior aspect. The eburnation of the trochlear surface is not well shown, nor the collar of osteophytes around each epicondyle.

In some instances there is a striking osteophytic outgrowth from the head of the bone, giving it a mushroom appearance. The greatest depth of this outgrowth is roughly 1·5 centimetres (five-eighths of an inch). (See Figure I.)

Eburnation of the surface for articulation with the capitulum of the humerus has occurred. The osteophytes have hidden by their outgrowth the articulating surfaces for the proximal radio-ulnar joint.

The evidence of the bones in which this extensive osteophytic development is found suggests a chronic

inflammatory process in the two joints extending over a comparatively long period of time.

The pathogenesis of osteoarthritis may be summed up in a quotation from Knagg's text-book on diseases of bone:

To those who have had much clinical experience of osteoarthritis two facts stand out prominently: (i) the not uncommon association of the condition with an injury

Figure III.

S.F. 20:94. The antero-lateral aspect of the distal half of a large right femur of an adult male aboriginal, depicting the marked structural alterations in bone architecture produced by a chronic inflammatory process of the nature of an osteomyelitis.

from which the onset of the trouble appears to date; (ii) the history of some infection or the presence of an infective focus in some part of the body.

It is considered possible that the infecting agent in these examples of severe osteoarthritis in Australian aborigines has been the *Treponema pallidum* of syphilis.

The Pathological Changes.

The pathological story unfolded by an examination of these 325 bone specimens of the skeleton proper of Australian aboriginal men, obtained from a limited geographical area of the south-eastern portion of the continent, presents a difficult problem for interpretation.

Several possibilities involving various disease processes present themselves. The investigator is forced to record impressions and to sift the meagre literature dealing with the diseases of aborigines since the arrival of Europeans. He must then correlate this information with recent observations of a scientific character on the only remaining available living material, the comparatively few aboriginal inhabitants of central and northern Australia.

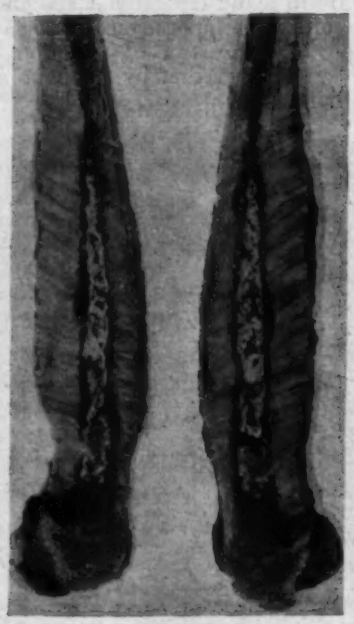


Figure IV.

S.F. 20:94. Vertical section through the distal half of this large male femur, showing gross changes due to long-continued osteomyelitis persisting from youth to adult age. Note marked cortical reaction, inspissated medullary contents or sequestrum and drainage cavity at distal extremity.

The task of attempting to fit disease labels to pathological bone specimens, without the possession of any scrap of clinical or authentic history, is one well calculated to make the boldest theorist pause. In this connexion the following quotation from Greig's "Clinical Observations on the Surgical Pathology of Bone" is very apposite:

To comprehend the full significance of the changes found in bone after injury or disease it is not enough to learn merely the cause of death or the nature of the disease. What is wanted is the full story of the individual with a detailed account of the course of the latest illness, as well as of the treatment adopted; for every one of these may modify the ultimate bony manifestations. Too frequently the information is brief and inadequate. Too often the story has to be built up from the specimen, and not the specimen correlated with a clinical history; yet there is no doubt which is the more instructive and the more valuable.

Bearing in mind this cautious reference to the difficulties ahead, I approached with great diffidence the diagnosis of the essential causal factor of the striking changes encountered in these bones.

Observation of these particular specimens over a period gradually reduced the original confused impressions to some semblance of order. The fractures are soundly healed, even if the apposition of the fractured ends is not in correct alignment.

FIGURE IVa.

S.F. 20 : 94. Posterior view of right adult male femur, showing massive reaction to chronic osteomyelitis, the extensive periosteal changes and the small round drainage cavity at the distal extremity.

Osteoarthritis is confined to two major joints, and has possibly been localized by injury. There is no other instance of any joint disturbances. It is, however, when the great majority of the bones which have been classified as pathological examples of periostitis and osteitis are under consideration that grave difficulties are encountered.

After somewhat prolonged examination the opinion has been gradually strengthened that in nearly all of them is present a definite and extensive degree of bone sclerosis. This is very pronounced in certain examples, and is in many instances accompanied by new bone formation of a denser character than the original. Both vertical and horizontal sections have been made to show the internal macroscopic bony structure, but it has not been found possible to prepare any microscopic slides. Röntgen ray photographs have failed, owing to the density of the sclerosis, to reveal any definition of the structural change which would assist in the diagnosis.

The evidence points to chronicity of an inflammatory process, which has given a sufficiently long period of time for the production by the bone of a

FIGURE V.

S.F. 20 : 97. The posterior aspect of the lower third of a male femur with gross enlargement caused by chronic osteomyelitis. Note circular drainage cavity. Vertical section shows similar changes to S.F. 20 : 94.

characteristic and unusual type of healing reaction. The two inflammatory conditions of periostitis and osteitis have been somewhat arbitrarily differentiated, as it is recognized that restriction of infection to the periosteum is evanescent in acute diseases and but temporary in chronic diseases.

There are some eighty odd specimens in which periostitis, either localized to small areas or generalized throughout the greater part of the bone, is found. They have been so classified because in these instances the inflammatory process, although in many cases extensive, is principally confined to the cortical portions of the bones and is of a uniform character, without any noticeable tendency

to destruction of bone. Occasional examples are seen in which the cortex has invaded the medullary area, with obliteration of the medullary cavity and the conversion of the cancellous structure, wholly or in part, into solid bone. Among a series all gradations of this remarkable invasive process may be observed, and the longitudinal area of bone involved is extensive.

Osteitis, of which there are also nearly eighty specimens, is regarded for the purposes of this investigation into aboriginal bone pathology, as a generalized inflammation of the whole bone. The specimens illustrating this condition are distinguished from those of periostitis by some amount of bone destruction, usually superficial in nature, associated with the universal sclerotic reaction.

Figure VI.

S.F. 20 : 87. The postero-external aspect of the middle third of a left male femur. There are marked changes due to osteitis, extensive thickening accompanied by superficial caries.

The two varieties of osteitis, rarefying osteitis or caries, and condensing osteitis or sclerosis, are well illustrated among these museum specimens. The latter predominates to a definite and unmistakable degree. The femora especially exhibit marked changes caused by some extensive and deep-seated inflammatory process. This has apparently been of low virulence and continued over a comparatively long period. The bone reaction has been of such an extent as to produce, in some instances, an extraordinary increase in size and density.

Although in the majority of specimens of osteitis it is observed that sclerosis, with increase in size and weight, is the essential pathological lesion, yet some of them give evidence of superficial caries with cavitation. This destructive change is practically confined to the exterior of the bone; it may have occurred subsequent to the generalized sclerotic process, or it may have gone on hand-in-hand with it, but attacking only the superficial layers of the bone.

Osteomyelitis.

I have been able to label only five specimens as osteomyelitis. In the absence of infection from without, this condition is considered to be confined to growing bone, and the definition is restricted to blood-borne infections. In two of these bones there is a peculiar correspondence to the specimens of septic osteomyelitis which are occasionally seen in surgical museums and which date back to the era preceding the introduction of anæsthetics and antiseptics. They are probably examples of a septic osteomyelitis persisting from youth into adult age. Their size and shape must excite interest and astonishment that the bone-forming capabilities of the individuals were so extensive.

Figure VII.

S.F. 20 : 87. Vertical section through the middle third of the left femur, showing marked changes due to osteitis. The sclerotic invasion has been complete, except for a small area of deep-seated caries having no communication with the exterior. This may have been a gummatous patch. Horizontal section at *A* shows complete sclerosis throughout the bone.

A detailed account of the several specimens in the museum catalogue will illustrate this description. In the first, the true nature of the underlying process was revealed only by vertical section of the bone.

Specimen S.F. 20 : 94.—Specimen S.F. 20 : 94 is an unusually large right femur of an adult male aboriginal, in the distal half of which pronounced pathological changes are found. This specimen is an outstanding example of the structural alterations in bone architecture which may be produced by a chronic inflammatory process. Its weight is 770 grammes (27¼ ounces), while the average separate weight of two normal aboriginal femurs in the museum is 424 grammes (15 ounces). (See Figures III, IV and IVa.)

Measurements taken in centimetres are as follows. The circumference at the widest portion, 11·5 centimetres above the articulating surface of the condyles, is 18·5 centimetres. That of a normal femur is 8·6 centimetres. The lateral and antero-posterior measurements taken by caliper at this same point were: lateral measurement, 5·9 centi-

metres; sagittal measurement, 5·6 centimetres. The normal lateral measurement is 2·7 centimetres, and the normal sagittal measurement 2·5 centimetres.

A careful examination of this bone reveals no evidence of any fracture in the lower third of the shaft.

The distal half of this femur is irregularly thickened anteriorly, with a gross generalized enlargement of this portion of the bone. There is some superficial flaking, and there are three superficial small carious cavities on the anterior aspect.

Posteriorly there is definite evidence of periosteal activity, with the formation of rough, sharp, bony outgrowths. These are of considerable extent, and can be traced in a less degree along the *linea aspera* to the trochanteric surfaces.

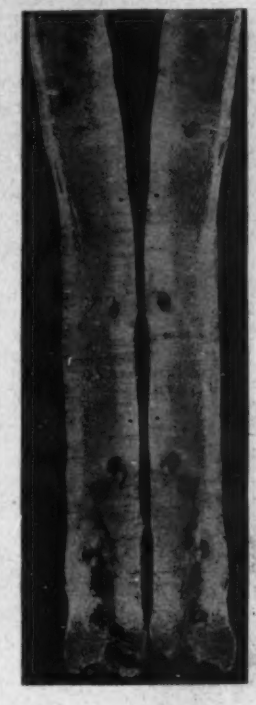
The most striking characteristics about this bone, however, are its great enlargement in the distal half, its solidity and its heaviness. The periosteal reaction gives only slight evidence that superficial caries with necrosis has at any time been prominent.

Continuous deposition of new bone caused by some chronic inflammatory process acting over a comparatively long period is the outstanding characteristic. The articular surfaces of the femur are affected to a minor degree. At the lower (distal) end of the bone, on its posterior aspect, between the condylar articular surfaces, is a small round drainage cavity, nine millimetres in width, which probably during life connected with the synovial cavity of the knee joint. A vertical section through this bone reveals changes of a somewhat surprising character, which are consistent only with the diagnosis of septic osteomyelitis in youth, persisting into adult life. The medullary cavity appears at first sight to be nearly filled in its distal half by a sequestrum, and to communicate at its lower end with a large drainage cavity leading posteriorly to the exterior. On further examination it is considered that this sequestrum is not a true bony one, but is possibly inspissated pus and medullary contents. The cortical walls of the medullary cavity are grossly thickened, the posterior wall measuring 2·1 centimetres, and the anterior wall 1·8 centimetres. This increase in thickness extends in a variable degree over a vertical distance of 22·5 centimetres. The medullary cavity has been considerably diminished in size, from 17 millimetres to an average of 13 millimetres, by the encroachment of its walls. In the distal articular end of the femur osteoarthritic changes are seen.

FIGURE VIII.

S.F. 19 : 26. Generalized osteitis of the tibia, showing loss of normal outline with carious destruction in the vicinity of the ankle joint, on the inner aspect of the bone *et cetera*.

SPECIMEN S.F. 20 : 97 (24).—In specimen S.F. 20 : 97 (24) similar changes, but not so gross or extensive in character, are seen in the distal half of a left male femur. A deep circular cavitation is present on the posterior aspect of the bone, about 2·0 centimetres above the distal articular surfaces. This connects with the medullary cavity, and measures 2·0 centimetres in length by 1·5 centimetres in breadth and 1·5 centimetres in depth. There is definite increase in the density and weight of the bone. (See Figure V.)



Vertical section reveals changes almost identical with those of the previous specimen, to a diminished degree.

Vertical sections have been made of all the bones in which considerable inflammatory reaction is seen, and in only three other examples have I considered that the diagnosis of osteomyelitis was reasonable. These specimens exhibit unusual changes in the tibia, and in contradistinction to those observed in the femur, they appear to be possibly of a secondary character.

FIGURE IX.

S.F. 19 : 26. Vertical section of a massive right male tibia, showing generalized osteitis with evidence of severe caries of the nature of an osteomyelitis in its distal fourth. It is difficult to determine whether this necrosis is gummatous breaking-through in the proximity of the ankle joint or a septic invasion from below. The sclerosis is the predominant process, and horizontal section at about the middle shows almost completely solid bone. (See Figure XXXI.)

SPECIMEN S.F. 19 : 26.—Specimen S.F. 19 : 26 is typical. This specimen illustrates in a pronounced degree a generalized thickening, solidity, increase in weight and loss of the normal outline in a massive right male tibia. In the shaft of the bone all the usual anatomical landmarks are absent. There is a uniform increase in size and rotundity, with varying degrees of roughness of surface. The inflammatory process has embraced the whole of the shaft and articular ends, and has produced also an extensive periosteal reaction. (See Figure VIII.)

Externally, one fair-sized necrotic area, 1·25 centimetres by 1·25 centimetres, exists in the centre of the bone, on its antero-internal aspect, and there are several small ones.

Apart from these occasional areas of caries, it is evident that sclerosis of the bone is the predominant characteristic. The greater portion of the articular surface for the ankle joint, however, especially on the inner aspect, has been broken down by a destructive process, which

FIGURE X.

S.F. 19 : 31. Vertical section of left male tibia, showing internal half-section. The sclerosis is pronounced, with patchy, apparently superficial caries in the lower quarter surrounded by sclerotic bone. This suggests gummatous breakdown. Horizontal section at *A*, see Figure XXVIII.

extends through to the posterior surface, with formation of bony bridges and a small sequestrum. Osteophytic

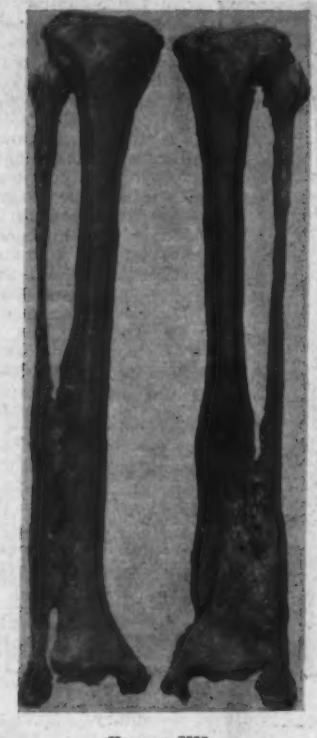


FIGURE XII.

S.F. 19 : 40. This unusual photograph shows an extensive bilateral infection uniting both tibiæ and fibulæ of the same individual. The condition appears to be an adhesive periostitis, but is accompanied by gross sclerosis of the two bones. Section has not been attempted, to avoid spoiling the specimens. There is no osteoporosis.

outgrowths have developed around the articulating surfaces forming the knee and ankle joints. On vertical section the medullary cavity is seen to have been obliterated throughout its entire length. In the proximal fourth its place has been taken by firm cancellous bone, and in the distal three-quarters by solid, ivory-like sclerotic bone with a narrow core of hard cancellous bone (see Figure IX).

In the distal fourth the one-time medullary cavity is obliterated by solid bone; but in the centre of this, caries with considerable necrosis has extended over a length of 8·7 centimetres. This area of destruction, which may be gummatous in nature, is surrounded by hard, solid, new bone, and when seen in section is comparatively superficial, although in the centre of the shaft. There is a

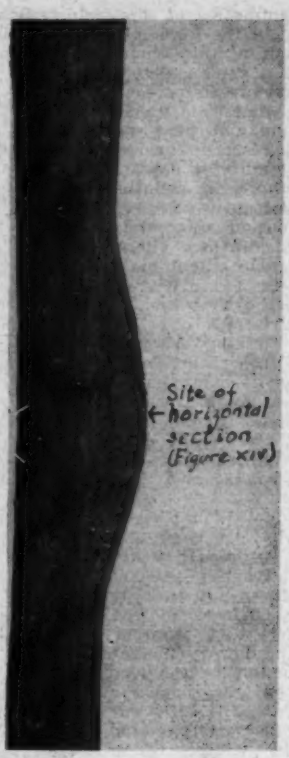


FIGURE XIII.

S.F. 20 : 85. Photograph of a periosteal node of the femur, about the centre of the shaft of a right male femur (see Figure XIV).

direct connexion between it and the abscess cavity in the ankle joint. It is possible that an invasion of septic organisms from below has occurred, and that this has spent itself without forming sequestra in the shaft or without impairing the solidity of the sclerotic framework.

FIGURE XIV.

S.F. 20 : 85. Horizontal section of the femur at site of the nodal invasion, with marked diminution of medullary cavity and sclerosis of the invading cortex (see Figure XIII). This illustration shows horizontal sections of three different femura. S.F. 20 : 96 is of a periosteal node in the upper third of a right femur. S.F. 20 : 85 is of a periosteal node in the middle third of a right femur, and S.F. 20 : 92 is of osteitis with sclerosis and superficial caries in the lower third of a right femur.

The appearance suggests that this invasion, of the character of osteomyelitis, may have been a secondary infection superimposed upon the original sclerotic reaction to some long-standing bone infection. A horizontal section is seen in Figure XXXI.

SPECIMEN S.F. 19 : 31.—Similar changes in almost every particular are found in specimen S.F. 19 : 31, except that the internal necrosis in the centre of the lower fourth of the bone is more superficial and has not forced its way to the exterior in the neighbourhood of the ankle joint. There is no sign of

medullary cavity, and it is difficult to explain this carious area, extending a longitudinal distance of seven centimetres and enclosed by sclerotic bone, unless the assumption of a gummatous breakdown is made (see Figures X and XI). X ray examination reveals a severe degree of sclerosis with only a limited amount of caries.

If these two specimens can be classified as osteomyelitis, they are certainly quite different from the two previously described specimens of the femur.

Figure XV.

S.F. 20 : 83. Vertical section through the lower third of a right male femur, showing extensive periosteal deposit without invasion of the medullary canal by sclerotic bone, but with noticeable increase of cancellous tissue.

Specimen S.F. 19 : 27, number 39.—In specimen S.F. 19 : 27, number 39, a generalized infection of the whole of the shaft of the tibia is found. This is especially pronounced on the posterior aspect and in the lower third. Numerous small superficial cavities give a worm-eaten appearance to the specimen. Caries with necrosis, and accompanied by rarefaction of the bone, has, from surface examination, apparently overshadowed any sclerotic reaction.

This tibia is comparatively light in weight. The normal outline of the shaft is lost, and the distal half evidences a continued destructive inflammatory process.

On vertical section the medullary cavity is seen to be present only in the proximal half of the bone. The cortical walls are sclerosed, and any rarefaction with caries is superficial. In the distal half obliteration of the medullary cavity and a greater development of sclerosis are seen; and the extensive caries is superficial. A secondary infection of the nature of osteomyelitis has spread from below into the sclerotic bone for a distance of 11 centimetres. No defined sequestrum is present, but great destruction has occurred in the lower eighth of the bone, with probable formation of an abscess cavity in the proximity of the ankle joint.

Periostitis.

The examples of periostitis found among these aboriginal bones are of great interest. They are most common in the femora, tibiæ, fibulæ and radii (see Figures XII, XIII, XIV and XV). In the femora the periostitis may be localized, nodal, or generalized, and typical specimens exhibit the characteristic changes.

Specimen S.F. 21 : 86.—In specimen S.F. 21 : 86 a localized periosteal reaction is seen about the middle of the shaft of a right femur, of an adult male, on its posterior surface. This has taken the form of a rough bony enlargement with sharp irregular edges. Its length is 3·7 centimetres and it is raised 1·0 centimetre from the *linea aspera*.

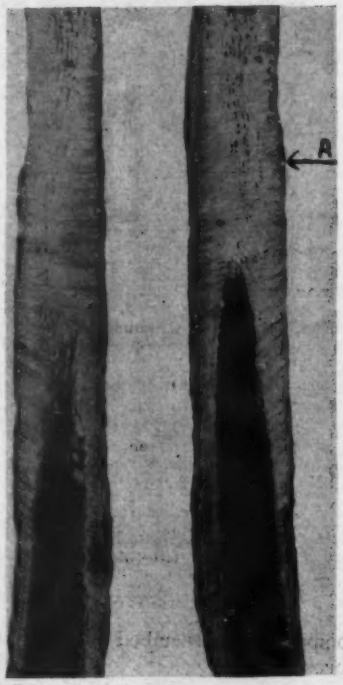


Figure XVI.

S.F. 20 : 86. Generalized periostitis of the femur with irregular thickening. This photograph of a vertical section shows marked invasion by sclerotic cortical bone. A horizontal section at *A* shows only a greatly diminished medullary cavity surrounded by sclerotic bone. One of the halves of the vertical section is too thin.

Specimen S.F. 20 : 85.—In specimen S.F. 20 : 85 the horizontal section is an excellent example of a periosteal node. It occurs on the inner aspect of the middle third of a right male femur, and extends over a vertical distance of 7·0 centimetres, being raised from the shaft about 1·25 centimetres. Horizontal section reveals completely solid bone encroaching on a medullary cavity reduced in diameter to 1·25 centimetres. (See Figures XIII and XIV.)

Specimen S.F. 20 : 86.—Specimen S.F. 20 : 86 is an example of generalized periostitis of the femur. There is irregular thickening over the proximal three-quarters of the shaft. Vertical and horizontal sections reveal invasion of the medulla in its middle third by dense cortical bone and solidified cancellous bone, with almost complete obliteration of the cavity (see Figure XVI).

In the case of the tibia there are gradations of the periosteal thickening, with the formation of cortical nodes at localized areas, and in extreme cases complete invasion of the bone.

Figure XVII.

S.F. 21 : 16. Massive periostitis of a left radius seen from the dorsal aspect. The bone is greatly enlarged in its distal two-thirds. A normal radius is shown for comparison (see section, Figure XVIII).

The two specimens described below are illustrative of the process.

Specimen S.F. 19 : 55.1.—In specimen S.F. 19 : 55.1 is seen pronounced thickening of a nodal character on the external surface of the upper third of the shaft of this tibia, with rough raised periosteal outgrowth on its posterior surface, extending for 5·0 centimetres (two inches). Vertical section reveals closure of the medullary canal in its proximal third, mainly by cancellous tissue, but at the site of the node there is a pronounced cortical invasion of solid bone.

Specimen S.F. 19 : 55.—In specimen S.F. 19 : 55 there is a generalized periosteal thickening of the proximal seven-eighths of the shaft, with rough periosteal outgrowth on the posterior surface to an extent of 7·5 centimetres. Vertical section reveals absence of the medullary cavity throughout the length of the bone. There is considerable cortical thickening, with gradual increasing invasion of the cancellous tissue filling this cavity, until in the centre of the bone, over a vertical length of 3·5 centimetres, it is replaced almost entirely by solid sclerotic bone.

Specimen S.F. 19 : 51.—Specimen S.F. 19 : 51, which is an unusually large and abnormally thickened tibia, on vertical section is found to bear a pronounced similarity to this specimen.

Specimen S.F. 19 : 52.—A further specimen of periostitis, specimen S.F. 19 : 52, on vertical section exhibits nodal formation with cortical increase in its upper fourth and invasion of the medullary cavity in its lower three-quarters. This is occluded mainly by sclerotic bone and partly by cancellous bone undergoing sclerosis. The course of the nutrient artery with accompanying sclerosis is plainly visible.

At the upper part of the lower fourth of the interior of the bone there are three coalesced cavities, which are irregular but clean-cut at base and edges. These are very suggestive of the remains of gummatous activity (see Figure XXX). There is no surface necrosis.

Periostitis of the Fibula. There are 18 examples of periostitis of the fibula.

Specimen S.F. 19 : 64.—A typical specimen of periostitis of the fibula is S.F. 19 : 64. In this is seen generalized periosteal enlargement of the distal three-quarters of the bone, with roughening of the margins of the comparatively deep groove on the postero-lateral surface. The increase in the size of this portion of the bone is to approximately twice normal, and is accompanied by increase of density and weight. Horizontal section at the junction of the middle and lower thirds reveals obliteration of the medullary cavity, mainly by ingrowth of solid bone from the periosteum, but also partly by thickening of the cancellous bone.

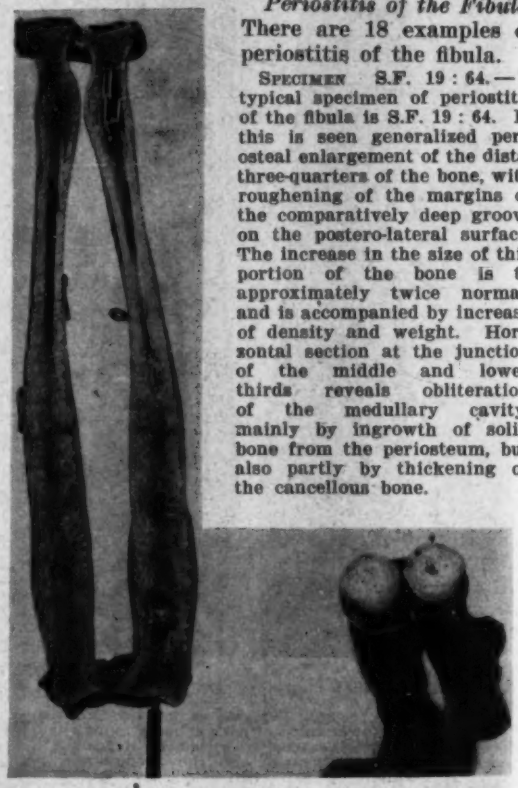


Figure XVIII.

S.F. 21 : 16. Vertical section of radius showing massive periostitis and obliteration of the medullary cavity over the distal half by very firm cancellous bone, which would appear to be gradually becoming converted into sclerotic bone. S.F. 20 : 32. Horizontal section of humerus, showing solid bone.

Periostitis of the Ulna.—There are many examples of periostitis of the ulna.

Specimen S.F. 21 : 44.—Specimen S.F. 21 : 44 is the type specimen of periostitis of the ulna. Generalized enlargement and thickening of the whole bone are visible. The special features are nodular enlargements, with irregularity and deformity of the normal bony outline of the shaft; also the presence of some sharp bony outgrowths. There is a minor degree of osteoarthritic change in the neighbourhood of the elbow joint. Horizontal section through a nodular enlargement reveals thickening of the cortex and closure of the medullary canal by firm cancellous tissue, with only a minor degree of cortical invasion.

Periostitis of the Humerus.—Periostitis of the humerus is represented by several examples in this collection.

Specimen S.F. 20 : 10.—Specimen S.F. 20 : 10, which is the right humerus of an adult male, is typical. There is uniform thickening of the distal three-quarters of the shaft, with loss of normal outline. Horizontal section in the middle of this bone, through a periosteal node, reveals an advanced stage of the general sclerotic process. The interior of the bone is solidified, and there is no medullary cavity. The cortex, with its dense sclerotic bone, has steadily encroached on the medullary space, which is centrally filled with a hard core of cancellous bone.

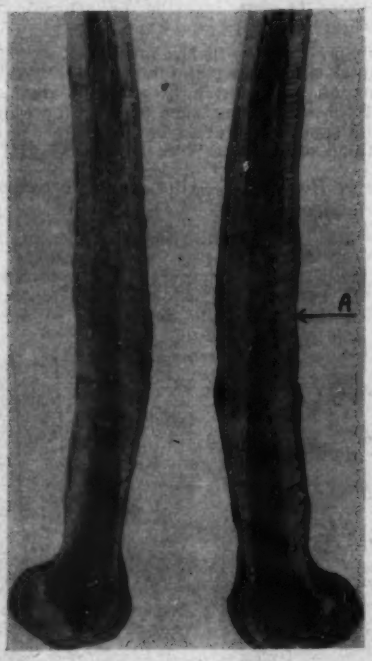


Figure XIX.

S.F. 20 : 89. Osteitis and coincident periostitis with irregularity of outline of a right adult female femur. On vertical section no medullary cavity is seen; it is either filled by cancellous tissue or, as at *A*, where a horizontal section has been made, by a mixture of cortical and cancellous bone.

Periostitis of the Radius.—There are numerous specimens showing the typical invasion of the medullary cavity by cortical ingrowth.

Specimen S.F. 21 : 16.—Specimen S.F. 21 : 16 is taken as an example of periostitis of the radius. In this particular bone is found a generalized smooth enlargement of the distal half of the shaft of the radius, with alteration of the normal outline and increased weight of the bone. The increase in size is to approximately twice normal. On vertical section the medullary cavity in the distal half of the bone is found to be absent, and is replaced by a solid mixed mass of cancellous bone and cortical ingrowth. The bone is generally thickened over all this area (see Figures XVII and XVIII).

Specimen S.F. 21 : 11.—Specimen S.F. 21 : 11 is another example of periostitis of the radius. Horizontal section at the junction of the middle and lower thirds reveals complete obliteration of the medullary cavity by solid bone, with no remains of cancellous tissue.

Osteitis.

The chief pathological condition is osteitis. This varies in the individual bones, and typical specimens will be described.

Osteitis of the Femur.—One example of osteitis of the femur is described.

Specimen S.F. 20 : 87.—In specimen S.F. 20 : 87 pronounced changes are seen in the middle third of the shaft of this left male femur, extending proximally into the upper third. There is loss of normal alignment of the shaft, accompanied by antero-posterior bowing in the middle third. It is possible that this was the site of a compound fracture. On the postero-external aspect of the shaft there is extensive cavitation with undermining, causing bony bridging. Thickening and deformity of the bone have taken place. Vertical section reveals a gross degree of sclerotic enlargement of the middle third of the bone, with almost complete obliteration of the medullary cavity over an area 19·0 centimetres in length by 5·9 centimetres in width at its widest part. There is a small area of caries. The sclerosis in the interior of the bone has successfully prevented any spread internally of the destructive activities of the superficial infection (see Figures VI and VII).

Osteitis of the Tibia.—One example of osteitis of the tibia is described.

Specimen S.F. 19 : 37.—Specimen S.F. 19 : 37 is an unusually large and heavy left tibia of an adult male aboriginal. At the junction of the upper and middle thirds, mainly on the posterior aspect, there are two areas of superficial necrosis extending over a vertical distance of 3·0 centimetres. Horizontal section at this point shows the infection to be wholly on the surface. The interior except for a small cancellous core, is filled with solid ivory-like bone.

A further horizontal section at the junction of the middle and lower thirds confirms this observation that the interior of the bone is almost entirely smooth, hard, sclerotic bone.

Osteitis of the Fibula.—There is one example described of osteitis of the fibula.

Specimen S.F. 19 : 35.—In specimen S.F. 19 : 35 pronounced inflammatory changes are seen in the distal seven-eighths of the bone, with increase in size to approximately three times normal, and increase in density. This is shown by the increase of weight. The normal shape and outline are altered. Except for two moderately sized carious cavities and several small ones, no evidence of osteoporosis exists. The final result has been predominantly a sclerosis of the whole of the distal seven-eighths of this fibula, with great periosteal reaction (see Figure XX). X ray examination reveals intense sclerosis, with small areas of rarefaction in the proximal third (see Figure XXI).

Figure XX.

S.F. 19 : 35. Osteitis of the greater portion of a left fibula. The view is taken of the anterior aspect and affords striking evidence of the change in size and shape of the bone. There is very little evidence of osteoporosis. Periosteal changes are pronounced.

Vertical section reveals that, except in the proximal eighth of this bone, the interior has been converted into dense solid bone, not showing any trace of medullary cavity. Just distal to the point where this cavity disappears there is a small round area of rarefaction connected with an external focus of caries (see Figure XXII).

FIGURE XXII.

Showing specimens S.F. 20 : 68 and S.F. 19 : 35, the former illustrating osteitis of the ulna with a large carious cavity in its distal fourth. Less than 2·5 centimetres above the superior margin a horizontal section reveals solid sclerotic bone (see Figure XXVIII). S.F. 19 : 35 is an incomplete vertical section of an osteitis of the fibula, showing gross sclerosis and one small carious cavity in the proximal fourth (see Figures XX and XXI).

Osteitis of the Ulna.—One example of osteitis of the ulna is described.

SPECIMEN S.F. 20 : 68.—In specimen S.F. 20 : 68 osteitis of the distal half of the ulna is seen. About the middle of the lower third of the shaft, over a longitudinal area of 3·75 centimetres (one and a half inches), there is extensive caries, which involves the greater portion of the bone. Severe necrosis has occurred over this limited area, with cavitation, tunnelling and overhanging bony ridges.

During life abscess formation with sinuses to the surface was probable. Horizontal section at the junction of the middle and lower thirds reveals, surprisingly, completely sclerosed solid bone throughout. The necrosis in the lower third, although extensive, is shown to be mainly superficial (see Figures XXII and XXVIII). It is not of the nature of osteomyelitis. The medullary canal has previously been solidified.

Osteitis of the Humerus.—Osteitis of the humerus is represented by two specimens.

SPECIMEN S.F. 20 : 32.—In specimen S.F. 20 : 32 the external three-quarters of the distal articular surface of the humerus is missing, as well as the greater portion of the head of the bone. There are signs of rarefying osteitis extending nearly the whole length of the bone, with caries and cavitation and definite periosteal reaction on the posterior surface. Vertical section reveals almost complete consolidation of the distal seven-eighths of this specimen by dense sclerotic bone. There are islands of cancellous bone. The caries with cavitation is almost entirely confined to the surface. Horizontal section in the middle of the shaft reveals solid bone with only traces of cancellous bone. In different specimens of osteitis varying degrees of this invasion by sclerotic bone are seen (see Figure XVIII).

SPECIMEN S.F. 20 : 35.—In specimen S.F. 20 : 35 vertical and horizontal sections of the humerus reveal complete consolidation of the distal half, with an area, 2·5 centimetres by 1·875 centimetres (one inch by three-quarters of an inch), of superficial caries on the external aspect of the lower third of the bone (see Figure XXVIII).

Osteitis of the Skull.—One example of osteitis of the skull is described.

SPECIMEN S.F. 20 : 57.—In specimen S.F. 20 : 57 severe osteitis of the frontal region is illustrated. There is extensive caries involving more than half of the anterior aspect of the bone. Shallow, depressed cavitations are present, with thickening and heaping-up of pericranium,

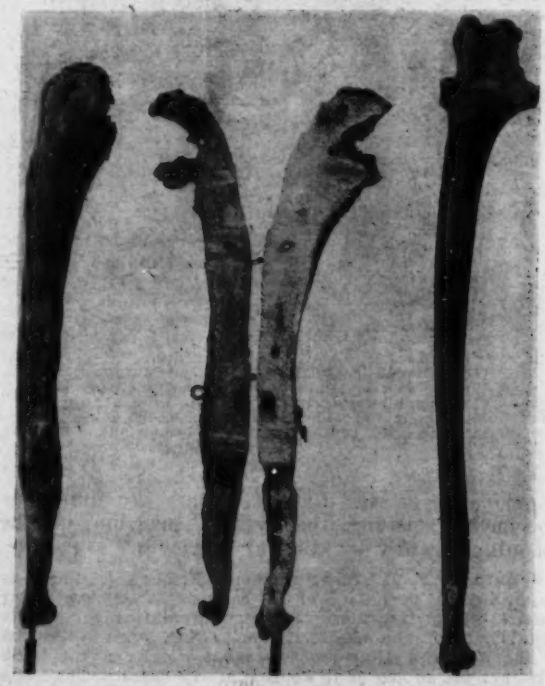


FIGURE XXIII.

Two ulnæ from one individual, S.F. 20 : 56 and S.F. 20 : 57. Both show marked changes due to osteitis; both show osteoporosis and osteosclerosis. S.F. 20 : 57 shows the external surface, and S.F. 20 : 56 the two halves of a vertical section. S.F. 20 : 49 shows osteoarthritic changes at the proximal extremity of a right ulna.

showing rough edges and solidification of bone. Both parietal bones give evidence of extensive osteitis with healed depressed areas, in which a minor degree of necrosis

is seen. Prominent ivory-like bossing is seen over the mid-frontal region. In other skulls similar features are found (see Figure XXV).

Comments on the Pathological Changes.

The pathological story unfolded by an examination of these 351 bone specimens of Australian aboriginal man, obtained from a limited geographical area of the south-eastern portion of the continent, presents, as has been previously mentioned, a difficult problem for interpretation.

There are nearly 200 specimens which have been classified as of periostitis or of osteitis. These present grave difficulties. In the great majority of them a definite degree of bone sclerosis is to be seen. This is very pronounced in certain examples, and, as previously stated, is in many instances accompanied by formation of new bone of a denser character than the original.

FIGURE XXV.

S.F. 20:57. Frontal region of skull, showing severe osteitis with caries, and bossing due to sclerosis.

What is the underlying causative factor which has brought about such marked changes in bone architecture? So far as our present knowledge of bone pathology extends, chronic inflammation of the long bones of the skeleton in man may be summarized as being caused by septic infection, tuberculosis, syphilis or yaws. The disease known as *osteitis deformans* (Paget's disease) has also to be considered, although, strictly speaking, this is not regarded as an inflammatory process. There is also the possibility that among the Australian aborigines there may have been observed the manifestations of an obscure inflammatory condition of bone which has not previously been recognized as a disease entity.

Further reference will be made to this later. On first consideration of the pathological changes in these aboriginal bones the obvious outstanding explanation would appear to be septic infection from without, by direct extension from the soft

FIGURE XXVI.

S.F. 19:36. Two halves of a vertical section, an excellent example of sclerosis of the tibia. Osteitis, with patchy small development of caries, but showing also the vigorous obliterating character of the sclerosis, is seen. The course of the nutrient artery can only just be distinguished.

tissues. It is easy to visualize the suitable environmental conditions of heat, dirt and flies, accompanied by ignorance of antiseptic or aseptic fundamentals.

The opening up of avenues of infection was and is of frequent occurrence. All available accounts, however, of the life and habits of the aborigines agree surprisingly upon the rapid and successful healing of severe wounds.

Horne and Aiston, in their book "Savage Life in Central Australia", state that "the black's resistance to inflammation is great compared with that of whites, although he more easily contracts other diseases".

In his book "Natives of Australia", N. W. Thomas makes the following statement:

> Like all savages, the Australian is singularly lacking in resistance to disease; in the matter of wounds, however, his viability is enormous.

"Wounds on the blacks heal with remarkable rapidity." ("Among Cannibals", by Lumholtz.)

Notwithstanding the presence of the numerous bone lesions due to septic infection which are found among the specimens in pathological museums dealing with examples from European communities, it is reasonably evident from the available data that sepsis is unlikely to have been the predominant causative factor in the pathological bone changes now under review.

Figure XXVIII.

Horizontal sections of three bones, giving marked evidence of the sclerosing type of osteitis. S.F. 19 : 31 is a section of a left male tibia. S.F. 20 : 68 is a section of an ulna previously described. S.F. 20 : 35 is a section of a left humerus.

Apart from the recorded observations on the living subject, the bones supply no evidence of large sequestra or of the presence of a violent destructive septic osteomyelitic process. All the evidence points to chronicity of infection accompanied by sclerotic reaction.

Figure XXIX.

S.F. 20 : 93. Showing the two halves of a vertical section of a left female femur. There are marked changes of osteitis, mainly of a sclerotic character, with small carious patches. Horizontal section at *A* shows a core of thickened cancellous tissue surrounded by a margin of encroaching solid bone.

The next consideration is tuberculous disease of bone. This is essentially a disease of childhood and of adolescence. Among the bones which are affected primarily are those of the vertebræ, the ends of the long bones *et cetera*. The shafts of the typical long bones are rarely affected by the tuberculous process.

In a table of the incidence of tuberculous disease in different bones and joints, the long bones of the skeleton are found to be affected to only a minor extent. In Knaggs's "The Inflammatory and Toxic Diseases of Bone" the following statistical data are given:

The femur, tibia and fibula combined are 3·5%. The scapula, ulna and radius 1·0%. The humerus 0·8%.

Tuberculosis of bone is more likely to be found in the neighbourhood of articulations, and usually produces a considerable amount of caries with bone destruction, unaccompanied, except in cases

Figure XXX.

S.F. 19 : 52. Sclerosing periostitis of the tibia, with clean-cut carious patches in the lower third suggestive of gummatous erosion.

of long-continued infection, by any well-defined amount of sclerotic change in the bone.

The pathological bone lesions seen in this collection at the Australian Institute of Anatomy do

not suggest tuberculosis. The areas of destruction are too circumscribed, they are mainly superficial, they are not near the joint surfaces, and the character of the bones is essentially adult. The reaction of the bones to the inflammatory stimulus is so definitely that of an intensive sclerosis that a diagnosis of tuberculosis does not appear to be feasible.

Examples of *osteitis deformans* are comparatively rare in pathological museums; and although the bone changes are in some respects comparable with those found in these specimens, yet these latter suggest a chronic inflammatory change rather than a disease process.

Figure XXXI.

S.F. 19 : 26. Osteitis of the tibia, with caries and necrosis at its lower end, as previously described. A horizontal section at *A* is also depicted.

It is stated that:

> . . . the bone changes in *osteitis deformans* are characterized by thickening, softening and deformity, and later by recalcification and hardening. The long bones are thickened, with surface irregularity and reduction in size of the medullary cavity. Bone absorption and deposition may proceed simultaneously, but ultimately ossification becomes more active and the enlarged bone is converted into hard, heavy and apparently strong osseous tissue. In the stage of softening and enlargement the bone is apt to become deformed.

The negative points in the diagnosis of Paget's disease are: first, the frequency of the occurrence of this definite pathological change, which has been described among these aboriginal bone specimens; secondly, the fact that while in *osteitis deformans* rarefactive and productive processes are proceeding together, in the examples of periostitis and osteitis under review, rarefaction is superficial and is possibly secondary in origin, gross deformity being consequently rare.

The cavities and cysts sometimes found in Paget's disease are not features of these bones. The invasion of the medullary cavity by cortical bone in the specimens under review is regular and uniform, without the thinning out into cavity formation more characteristic of *osteitis deformans*.

By a process of elimination the diagnosis is concentrated upon some form of spirochætal infection, either acquired syphilis, endemic syphilis, yaws or some unknown pathological entity peculiar to the Australian race.

To show that this latter suggestion is not fantastic, I may refer to the only clinical report available to me of an aboriginal patient suffering from the bone lesions of arthritis, osteitis and periostitis. The authors, A. Breinl and M. J. Holmes, after describing the appearance of the right tibia and both ulnæ, are forced to make the unusual diagnosis of a "disease *sui generis*". Their description, which is embodied in a medical report on the Northern Territory, gives a series of clinical signs which would fit quite accurately many of the bone lesions seen in specimens in the museum of the institute.

The only other available clinical description is that by a non-medical observer, Sturt, the explorer. In describing the appearance of the natives in the area between the junctions of the Murrumbidgee and the Darling with the Murray, he makes the following statement:

> The most loathsome diseases prevailed amongst them. Syphilis raged among them with fearful violence.

The exact diagnosis may be discounted; but the area from which the bones in this institute were obtained corresponds roughly with that of Sturt's description. There is a definite possibility that these pathological bone lesions of such an unusual character have been caused by the ravages of some

Figure XXXII.

A lateral view of two femurs from an adolescent (note absence of epiphyses) who has probably been the victim of some nutritional defect, causing such a profound change in the shape of the bones.

disease process not known to Europeans; but it is more likely that they are spirochætal in origin.

A description was given in *The Transactions of the Royal Society of Tropical Medicine and Hygiene* of June, 1937, by E. H. Hudson, of *bejel*, the

endemic syphilis of the Euphrates Arab. This is a form of non-venereal syphilis which is endemic among the Arab population of part of the Syrian desert. It is regarded as a disease of childhood and not associated with sexual infection. Lesions may involve the skin and the bones. It occurs in a region far from the accepted geographical definition of yaws.

The author states that there is doubtless general agreement that there exists among the lowly populations of large areas of the world a tremendous amount of disease due to the *Treponema pallidum*. This "indigenous syphilis" has been recognized in many places and given many descriptive names. It

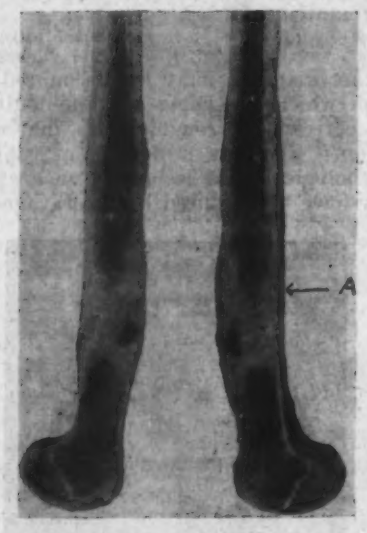


Figure XXXIII.

S.F. 20 : 92. Osteitis and periostitis in the distal half of the femur, showing obliteration of the medullary cavity. Subsequent horizontal section at *A* shows almost entirely solid bone throughout; only a thin core of cancellous tissue remains.

was possibly endemic in Europe long before the discovery of America and the recognition of venereal syphilis.

I have given only a brief reference to this account of a disease which might possibly explain the condition of the Australian aborigines as observed by Sturt.

Lastly, there is the alternative diagnosis between venereal syphilis and non-venereal yaws. Both are spirochætal infections, and in the tertiary stage both may produce severe bone lesions.

It has been found impossible to obtain bones from patients who have died from authenticated yaws, in which pathological lesions could be seen, although the attempt has been made in Papua and elsewhere. Also, owing to the double effect of intensive modern treatment, combined with the result of a gradual immunization of the populations, European bone specimens in which advanced syphilitic changes can be seen are uncommon in Australian pathological museums.

The changes in the aboriginal specimens under consideration have, then, to be diagnosed on their pathological appearance, so far as this conforms with accepted text-book descriptions of disease processes in bone. The differential diagnosis between yaws and syphilis as the causative disease factor responsible for the changes observed in these aboriginal bones is therefore necessarily a difficult one. It is a matter of some controversy. During the past two years a forceful, well-informed and scientific attempt has been made to prove several cardinal assertions relating to the prevalence of the two diseases, syphilis and yaws, among the Australian aborigines (*vide* C. J. Hackett, The Medical Journal of Australia, May 30, 1936).

The assertion which concerns this present article is the following:

> The numerous aboriginal bones from southern and eastern areas of Australia, now in Australian museums, show yaws lesions.

Yaws has been known to the medical profession for at least one hundred years, as is shown by the prize essay, "Observations on Yaws", written by James Maxwell in 1839. In this the differential diagnosis between syphilis and yaws is given, and the bone changes caused by yaws are mentioned. The exact pathology of yaws, however, especially of its bone lesions, is still obscure. This is probably because, owing to its essentially tropical character, the scientific investigations have been spasmodic and few in numbers. Manson states that "yaws is practically confined to the tropics and sub-tropics, and even there is absent at high altitudes". In his view, the diseases of yaws and syphilis are distinct manifestations by two different forms of spirochætes. He particularly instances the absence of endarteritis and of gummata in yaws.

Although it has been stated by several writers that the bone lesions of yaws are severe in character (*vide* Spittel, in his publication "Frambœsia Tropica", and others), yet text-books on pathology rarely mention them.

As recently as 1936, in a discussion concerning Hackett's paper, "Boomerang Legs and Yaws in Australian Aborigines", at a meeting of the Royal Society of Tropical Medicine and Hygiene in London, the following statements were made: "Just how common bony lesions are in yaws we do not know", and "So much of our knowledge concerning yaws lacks exactitude".

It is obvious that the bone pathology of yaws is as yet obscure. Owing to the curability of yaws and the absence of any pronounced effect on the general health, authenticated pathological specimens must be very rare.

Even with the active assistance of the Commonwealth Department of Health I have been unable to obtain one bone specimen of yaws to compare with the bone lesions at present under review.

The following is one of the best brief modern descriptions of the disease, and strikingly illustrates

the difficulties of diagnosis. Lawford Knaggs refers to the lesions of yaws as follows:

> It will be readily appreciated that this controversy as to the true character of yaws introduces difficulty into an attempt to describe the features of its osseous lesions, and this difficulty is increased by the absence or rarity of well authenticated examples in the museums of this country [Great Britain]. In yaws as in syphilis the bones are affected in the tertiary stage.

He quotes Bahr's description of the bone lesions of yaws seen in Ceylon, acute periostitis, chronic periostitis, osteitis and epiphysitis.

He also describes twenty bone lesions of yaws investigated by means of X rays in Manila, in which multiplicity and rarefaction were predominant features.

Having been granted permission to examine the bone specimens in the museum of the Royal College of Surgeons, Knaggs describes several bones, tibiæ and fibulæ from New Guinea.

> All of them show osseous periostitic nodes, and some are quite indistinguishable from syphilis. But others present a curious pitted appearance on the surface of the node, as if a secondary caries had been going on in a considerable number of separate places. The pitted condition is quite different from the usual appearance seen in syphilitic affections of the long bones.

This description in brief would apply reasonably accurately to many of the pathological specimens of aboriginal bones in our museum.

It is very interesting to note the official description of these particular New Guinea bones in the Royal College of Surgeons museum catalogue, in which the question is summed up as follows:

> If the lesions are not regarded as due to syphilis, they can only be ascribed to a disease hitherto not met with, which produces similar osseous changes.

Yaws (*frambœsia tropica*) is a disease met with in tropical countries, especially the west coast of Central Africa, where it is supposed to have originated, the West Indies, Ceylon, the Malay Archipelago, certain Pacific islands *et cetera*. For more than one hundred years there has been confusion between yaws and syphilis among medical observers. At the present time opinion would appear to have hardened into the belief that yaws is not syphilis, but a separate disease. Its causal organism is also a spirochæte. Infection is produced by contact; the manifestations, which are mainly cutaneous, are chiefly observed in young children; and yaws is not regarded as a venereal disease.

Although yaws has been diagnosed as a common disease in northern Australia, Papua, Fiji and elsewhere, it has not been possible for this institute to obtain one undoubted pathological bone specimen. The conclusion is either that pathological bone lesions of yaws are impossible to obtain in the areas mentioned, or that they do not exist there.

Even if the geographical tropics are regarded as a somewhat elastic land definition, the particular area of Australia from which many of the bones in this museum were obtained—the Barham district in New South Wales near the River Murray—could not be regarded as part of them.

The continent of Australia contains a variety of climatic conditions, ranging from tropical through subtropical to temperate. A generalization, such as has been made by Hackett, that an undoubtedly accepted tropical disease such as yaws could flourish among a native population in such a temperate climate as that of the south-eastern corner of Australia, requires to be supported by more facts than have so far been presented by him before acceptance could be justified.

Whatever the present state of the aborigines is in northern central and northern Australia, where yaws is an accepted disease entity and is possibly increasing, it is a very far cry to postulate similar conditions one hundred years ago in New South Wales, Victoria and South Australia.

In my opinion a more feasible theory is that yaws has been originally introduced to the northern tropical parts of Australia by Malay contact from the islands of the East Indian archipelago; that it has spread, by contact especially, among children; that it has increased as tribal boundaries have been broken down by the impact of European civilization; and that it has been definitely limited in its spread by geographical climatic boundaries.

Yaws, being a spirochætal disease, may have conferred an immunity against syphilis among the tribes in which it has been endemic. A similar phenomenon was observed in Fiji (*vide* the report by C. W. Daniels).

On the other hand, syphilis among the Australian aborigines has probably been almost entirely a venereal infection. It has been introduced to the tribes by the male Europeans, and has spread wherever the white and the black have lived in close proximity and wherever climatic conditions have been eminently suitable for European colonization.

It is my opinion that when British settlement commenced in Australia the aborigine had conquered his environment and was a healthy animal, comparable in physical fitness with the animals around him. Contact with European civilization and disease destroyed him.

A careful consideration of the arguments advanced by Hackett to support his contention, that when the Australian aborigines were first discovered by Europeans yaws was present in all native communities, and that subsequent descriptions by medical and non-medical writers of syphilis and venereal disease were really descriptions of yaws, does not tend towards confirmation of this theory.

The detailed evidence in recent times of observers such as J. B. Cleland, of Adelaide, and H. Basedow, who describe and differentiate both diseases, cannot be lightly controverted. The former describes aboriginal bones found in South Australia, near the River Murray, affected with lesions of arthritis, periostitis and osteitis.

These are the same typical lesions as are found in our specimens; and Cleland describes their appearance as "strongly suggestive of the ravages of syphilis". He also states that no trace of yaws

has ever been found among the natives of South Australia.

Basedow describes lesions of syphilis among the aborigines, in a description comparable with those of the writers of the Middle Ages in Europe. "A sad chapter", he states, "could for instance be written upon the havoc which has been wrought by the imported scourge of syphilis among the tribes."

Hackett states that among the aborigines of the Northern Territory of Australia today syphilis is unknown, while yaws is frequent. It is important to compare this statement, which is one of the corner stones of Hackett's argument regarding yaws, with an official report.

Breinl and Holmes, in *The Bulletin of the Northern Territory*, Number 15, of December, 1915, make the following statement:

> The natives of the Northern Territory who had not to any extent come in contact with whites are of excellent physique and well nourished. Generally speaking, the health of the natives of the Northern Territory is excellent when compared with that of natives of other tropical countries. It is evident from our observations that the natives of the Northern Territory are in their natural state very healthy. Those, on the other hand, who have come into contact with the new settlers rapidly fall victims to the diseases which have been introduced with the settlement.
>
> Syphilis is not uncommon among the tribes, but only amongst those who have come in contact with whites and Chinamen. The introduction of syphilis is very serious, as syphilis affects natives far more seriously than other races amongst whom syphilis has been present for centuries.

In my opinion, this report is an epitome of the medical history of the Australian aboriginal race in the whole of its one hundred and fifty years of struggle, adversity and destruction since European occupation.

Holmes, in *The Bulletin of the Northern Territory*, Number 6, May, 1913, makes the following statement:

> As regards syphilis and gonorrhœa, both these diseases are very prevalent. Yaws is prevalent in various localities. In ten years at Darwin Hospital, from 1903 to 1912, forty-one cases of syphilis were treated, two only of yaws, and forty-four of *granuloma pudendi*.

As regards the bones themselves, I am strongly of the opinion that, while bearing in mind the possibility of some indigenous disease, we are dealing with the gross bone lesions of syphilis in a non-immunized and untreated native community.

Owing to the rarity of specimens and descriptions, I find it impossible to obtain a clear mental picture of the bone changes in yaws. The outstanding difference as regards our museum specimens would appear to be the gross general sclerosis in the latter, the frequency of the periosteal nodal type of lesion, and the comparative absence of the areas of rarefaction which are described in yaws. The presence of the definite periosteal proliferation and the pronounced cortical thickening are also distinguishing features as distinct from the bone lesions of yaws.

As regard some of the pathological features of the bones themselves, it is recognized that these may not completely conform to modern text-book descriptions of syphilis of bone. I think it must be admitted that human pathology, with its text-books and museums, is a comparatively modern science. The bone lesions of syphilis in Europeans have probably changed considerably in severity during the past one hundred years. Two factors have been at work to make the tertiary bone manifestations less severe: firstly, the gradual immunization of the population, and secondly, the improved methods of treatment of this disease. It is reasonable to assume, therefore, that syphilitic European bone specimens seen in pathological museums today do not correspond wholly in type with those from non-immunized, untreated aborigines.

Among the 165 specimens which I have placed in one or other of the categories of osteitis, periostitis and osteomyelitis, there are fifteen in which multiple superficial necrotic lesions are found. These occur among the tibiæ, humeri, ulnæ and scapulæ. They are classified as examples of osteitis with osteoporosis, and are distinguished by the presence of numerous superficial excavated depressions with undermined margins. The size of the depressions is variable in any one specimen, but the superficial destructive process is definite.

These areas of bone necrosis may possibly have been wholly caused by a pyogenic infection; but the appearance is more suggestive of a primary chronic inflammatory process breaking down the resistance of the bone to a secondary sepsis. On the other hand, it is more in conformity with my theories to assume that they are probably due to the severity of a syphilitic infection in a native population. There may have been a combination of these factors.

An interesting point is that, except among the flat bones, such as the scapulæ, the surface necrosis is accompanied by a great degree of internal osteosclerosis, which is most pronounced in the tibiæ.

The most striking single pathological curiosity about these aboriginal bones is the complete solidity which is sometimes evident on horizontal section. The bone is smooth, hard and consistently solid throughout. In the examples of periostitis in which this phenomenon occurs one is inevitably forced back upon a diagnosis of syphilis. Whether the virus has originated this change in the medulla or in the periosteum, the result is the same.

Once again it must be emphasized that sclerosis is the predominant feature in these bones, and sclerosis of an extent and density which are rarely seen. Many of the specimens must be unique in this respect, and can be regarded only as pathological rarities.

W. Quarry Wood, in an examination of 236 tibiæ of Australian aborigines, mainly from the Northern Territory, found very few examples of disease. In two specimens, however, undoubted evidence was found of *arthritis deformans* of the knee joint. In

one pair of bones signs of advanced syphilitic disease were found.

As a concluding argument in favour of the diagnosis of syphilis, it is interesting to make two final quotations:

> The osteosclerosis in syphilis is not limited to the new bone or to the surface. The fibrous element within the bone atrophies to some extent and is replaced by bone; the bone cells contract, withdraw their processes, the Haversian canals and the canaliculi are diminished in calibre, and the bone throughout becomes hard, close, dense and comparatively anæmic. Diseases characterized by restriction of the circulation (and typical of these may be cited *endarteritis obliterans syphilitica*) are accompanied by osteosclerosis and increased density of bone. (Greig's "Clinical Observations on the Surgical Pathology of Bone.")

It is considered that the very extensive periosteal reaction which is seen both in specimens labelled periostitis and in those considered to be of osteitis, and the pronounced sclerotic nature of the final stage of the disease process, are sufficient evidence to justify a probable diagnosis of syphilis, even if this is not the whole story.

The following quotation is of importance in this regard:

> Syphilis of bones affects the diaphysis rather than the articular ends (involvement of joints very seldom occurs); sclerosis is much more common than rarefaction. The presence of spirochætes seems to stimulate the osteoblasts to great activity, and an abundant formation of new bone is laid down; (i) on the surface the process may be uniform; (ii) on the walls of the Haversian canals the shaft of the bone may become extremely heavy and dense, with obliteration of the medullary cavity. (Extract from "Surgical Pathology", by Boyd.)

Syphilis of the long bones of the skeleton is of the nature of a diffuse osteoperiostitis.

> Bone-marrow is one of the chief seats of the syphilitic virus, and gives the most constantly positive results of artificial inoculation. (Lawford Knaggs.)

In its simplest form the syphilitic node is a purely periosteal affection; but the inflammatory process soon begins to attack the bone itself and spread into it. First, the compact tissue underlying the node is rarefied; then the adjacent cancellous tissue is involved; lastly, the medulla of the central cavity is affected; and in due time the rarefaction gives place to sclerosing osteitis. Sections through bones that are subjects of osseous nodes will reveal in some the condition to be superficial to the compact bone. In others an area of sclerosis will be found occupying the compact and adjacent cancellous tissue; and in a few the medullary cavity, in addition, will be completely filled at the level of the node by a mass of dense bone. These three stages are well shown in the specimens in the museum of the institute.

The destruction of the true original character of the bones, without persisting rarefaction, but with slow ossification of the newgrowth causing a gradual obliteration of the medullary cavity, the extreme heaviness of the bones, with the irregular thickenings from coincident periostitis, all these features, in my opinion, point to venereal syphilis as being the most probable factor in this remarkable change in bone structure and architecture.

The ultimate diagnosis must therefore, I consider, rest between venereal syphilis, an endemic type of non-venereal syphilis, or some indigenous disease of which we have no medical knowledge, which also produces these striking pathological lesions in bone.

The first alternative is, after full consideration of the historical, geographical and medical information available, regarded as the most likely.

Bibliography.

L. Knaggs: "The Inflammatory and Toxic Diseases of Bone".
D. M. Greig: "Clinical Observations on the Surgical Pathology of Bone".
T. Jones: "Diseases of the Bones".
P. Manson: "Tropical Diseases".
The New Sydenham Society: "Selected Essays", Volume LI.
T. H. Green: "Manual of Pathology".
W. Boyd: "Surgical Pathology".
Beattie and Dickson: "Text Book of Pathology".
R. Muir: "Text Book of Pathology".
R. L. Spittel: "Framboesia Tropica".
Mercer: "Orthopædic Surgery".
E. H. Hudson: *Transactions of the Royal Society of Tropical Medicine and Hygiene.*
The British Medical Association Committee: Report, "Causation and Treatment of Arthritis and Allied Conditions".
J. B. Cleland: "Disease among the Australian Aborigines", *The Journal of Tropical Medicine and Hygiene*, Volume XXXI, March 1 and 15, 1928, pages 53 and 65.
H. Basedow: "Diseases of the Australian Aborigines", *The Journal of Tropical Medicine and Hygiene*, Volume XXXV, June 15 to September 15, 1932, pages 177-273.
C. J. Hackett: "A Critical Survey of Some References to Syphilis and Yaws among the Australian Aborigines", The Medical Journal of Australia, Volume I, May 30, 1936, page 733.
C. J. Hackett: "Boomerang Legs and Yaws in Australian Aborigines", *Transactions of the Royal Society of Tropical Medicine and Hygiene*, Monograph I, 1936.
A. Breinl and M. J. Holmes: *The Bulletin of the Northern Territory*, Number 15, December, 1915.
M. J. Holmes: *The Bulletin of the Northern Territory*, Number 6, 1913.
Spencer and Gillen: "Across Australia".
Spencer and Gillen: "Native Tribes of Central Australia".
N. W. Thomas: "Natives of Australia".
Horne and Aiston: "Savage Life in Central Australia".
A. W. Howitt: "Native Tribes of South East Australia.
E. M. Curr: "The Australian Race".
R. Brough Smyth: "The Aborigines of Victoria".
Lumholtz: "Among Cannibals".
Report of the Select Committee of the House of Commons on Aborigines (British Settlements), 1836.
W. Q. Wood: "The Tibia of the Australian Aborigine", *Journal of Anatomy*, 1920, page 232.

THE TREATMENT OF PNEUMONIA.[1]

By A. Holmes à Court, M.D. (Sydney), F.R.C.P. (London),

Honorary Physician, Sydney Hospital; Honorary Consulting Physician, Royal Hospital for Women, Sydney.

The problem of pneumonia remains a matter of major importance in the practice of medicine; the difficulties of management and successful treatment are universally present and frequently confront the practitioner. When asked by your committee to discuss the modern treatment of pneumonia, I felt considerable diffidence in undertaking such a task; but, having had opportunities for studying recent work in this subject abroad, I regarded it as a duty to attempt to do so, however unworthily. A review

[1] A post-graduate lecture, delivered at Sydney on April 4, 1938.

of the literature of modern methods of dealing with this common disease and personal observation gained during a recent visit to America have prompted me to put before you some of the relevant facts of the present-day knowledge.

Post-graduate lectures are in most instances eulogies of the methods of treatment employed and records of successful results achieved in our larger hospitals. In the present instance, however, it is my purpose to show you how far we in this community lag behind the world in this subject, and to endeavour to arouse a lively interest in the problem of modern treatment, so that deficiencies may be made apparent with the object of ultimate improvement in our methods.

Described by the older authors as "Captain of the men of Death", the disease pneumonia continues to take grievous toll of human life, accounting in some communities for approximately one death in every fourteen from all causes.[1]

The opinion is current that in this country pneumonia is probably a less lethal disease than is commonly the case in other parts of the world, for example, the United States of America, possibly because of the comparatively good hygienic and dietetic conditions of the population generally, and of climatic conditions, or because of variation in the degree of virulence of the organism in particular outbreaks and in varying seasons.

Reference to the vital statistics for the State of New South Wales for the five-year period 1932 to 1936 (inclusive) shows that of the total deaths registered, 6·4% were attributed to pneumonia in some form; that is, approximately one death in every fifteen to sixteen.

Excluding bronchopneumonia and pneumonia unspecified, of the total deaths registered in New South Wales in 1936 (the last year for which statistics are available) lobar pneumonia is assigned as having been the cause of 761, that is, 3·21% of the total deaths. The average number of deaths *per annum* from this disease over the five-year period is found to be 690. It would seem likely, therefore, that there is no vast difference between the figures for this country and elsewhere. It is our duty to be informed of the recent advances in dealing with this disease, and to attempt to apply them as well as the means at our disposal permit.

In this discussion of the treatment of the group of infective processes of the respiratory organs classified as pneumonia, it is unnecessary to allude in detail to the accepted descriptions of lobar pneumonia and bronchopneumonia of the text-books, of which the details are all too familiar. The term "pneumonia" has come to be applied to any inflammatory process of the lung producing consolidation; and it is of importance to concentrate upon the nature of the infective agent rather than to discriminate too closely in the distribution of pathological changes in the respiratory tree. Hence a distinction too rigid need not be drawn between lobar pneumona and bronchopneumonia of adults and the bronchopneumonia of children.

Apart from the classical types of croupous or lobar pneumonia, frankly infectious by pneumococci of the several types, it is usual to recognize clinically streptococcal bronchopneumonia and the condition clinically familiar as "pneumonic influenza", now believed to be a virus disease.

It is important to realize that the secondary pneumonias, following acute infective diseases of children, measles, pertussis *et cetera*, are in a large proportion of cases due to superimposed pneumococcal infections.

In a recent review of pneumococcal infections, Dr. Jesse Bullowa,[2] of New York, has stated that probably 85% of primary pneumonic diseases of adults and at least 54% of the pneumonias of children are due to infection by pneumococci. The importance of these facts becomes manifest with the ability to distinguish the several types of pneumococci causing disease and the advent of efficient serum therapy, which promises to place pneumonia in the category of controlled diseases in the not far distant future.

Types of Pneumococci.

Including the well-known Types I, II and III, 32 recognizable types of pneumococci have been separated, distinguished by their agglutination reactions in homologous sera. The method of typing these organisms is comparatively simple if the Neufeld technique is used, provided the appropriate test sera are available.

The incidence of infection by organisms of the several types in a large series of cases has been

Table I.

Return showing Deaths from Pneumonia in New South Wales during the Five Years 1932 to 1936 inclusive and Percentage of Deaths from Pneumonia to Total Deaths.

Year.	Deaths due to Pneumonia.									Total Deaths from all Causes.			Percentage of Deaths from Pneumonia to Deaths from all Causes.								
	Broncho-pneumonia.			Lobar Pneumonia.			Pneumonia Unspecified.						Broncho-pneumonia.			Lobar Pneumonia.			Pneumonia Unspecified.		
	Male.	Female.	Total.	Male.	Female.	Total.	Male.	Female.	Total.	Male.	Female.	Total.	Male.	Female.	Total.	Male.	Female.	Total.	Male.	Female.	Total.
1932 ..	272	198	470	295	209	504	150	106	256	12,110	9,247	21,357	2·25	2·14	2·20	2·44	2·26	2·35	1·24	1·14	1·19
1933 ..	300	246	546	322	232	554	173	110	283	12,727	9,595	22,322	2·35	2·56	2·45	2·53	2·42	2·48	1·36	1·15	1·27
1934 ..	335	294	629	348	246	594	175	124	299	13,173	10,301	23,474	2·54	2·85	2·68	2·64	2·39	2·53	1·32	1·20	1·24
1935 ..	348	307	655	467	272	739	182	131	313	13,891	10,656	24,547	2·50	2·88	2·67	3·37	2·55	3·01	1·31	1·23	1·27
1936 ..	342	301	643	462	299	761	189	134	323	13,618	10,758	24,376	2·51	2·79	2·64	3·39	2·78	3·12	1·39	1·25	1·32

ILLUSTRATIONS TO THE ARTICLE BY DR. CHARLES V. MACKAY.

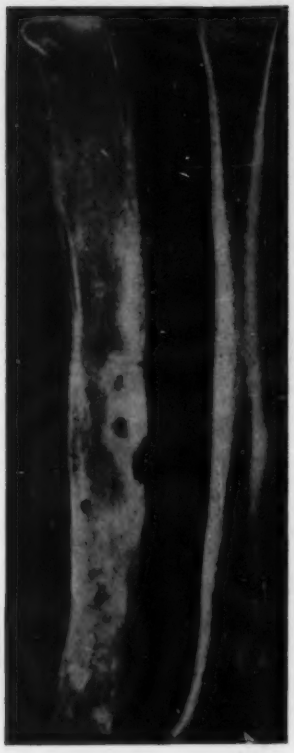


FIGURE XI.

S.F. 19:31. X ray photograph of vertical section of left male tibia, showing external half-section contrasted with a normal tibia. The absence of medullary cavity and the sclerosis causing loss of definition are well shown. A horizontal section in the lower third has shown solid bone.

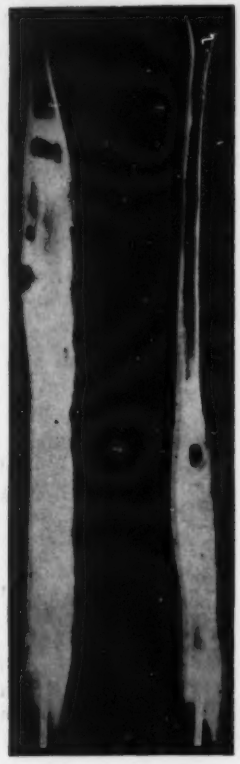


FIGURE XXI.

S.F. 19:28 and 35. An X ray photograph of two examples of osteitis of the fibula with marked sclerosis, impermeable to the rays, and patchy osteoporosis with caries. The contrast with normal unaffected bone is pronounced.

FIGURE XXIV.

S.F. 19:32 and S.F. 19:6. X ray photographs of osteitis of the clavicles. The former (left) shows sclerosis impenetrable to the rays, and the latter patchy osteoporosis with sclerosis.

FIGURE XXVII.

S.F. 19:19. X ray photograph of osteitis of the scapula, showing osteoporosis with surrounding sclerosis.

ILLUSTRATIONS TO THE ARTICLE BY DR. REGINALD WEBSTER.

FIGURE XX.
Dermoid cyst between the lobes of the cerebellum: Angulation of brain stem: Dilatation of lateral ventricle. Left half of brain.

FIGURE XXI.
Right half of brain. Depression on the medial aspect of the right lateral lobe of the cerebellum produced by dermoid cyst.

ILLUSTRATIONS TO THE ARTICLE BY DR. OSWALD J. ELLIS.

FIGURE I.
Showing the radiographic appearance ten minutes after the injection of "Uroselectan B".

FIGURE II.
Pyelogram of the upper (left) kidney.

FIGURE III.
Pyelogram of the lower (right) kidney.

worked out by Cecil[3] and other observers in certain regions of America. It will be observed that infections of Types I, II, III, VIII, V and VII accounted for 84% of cases of pneumonia, Types I, II and VII alone accounted for nearly 60%.

The importance of these observations lies in the fact that efficient sera have been prepared for most of the common types of infection. At the Harlem Hospital, in New York, it was found that sera were in use for infections by Types I, II, VII, V, VIII, XIV, IX, XVIII, VI, XIX, XII and XVII. The routine investigation and treatment employed are worthy of careful attention. As soon as the diagnosis of pneumonia has been established on clinical grounds, corroborated if necessary by radiography, efforts are at once made to obtain the pneumococcus for classification.

The organisms may be obtained from sputum and from specimens obtained by lung puncture and blood culture, or from other sources. In young children who tend to swallow the sputum, examination of gastric contents may be successful. Rapid typing is carried out by agglutination methods, controlled by mouse inoculation and culture.

Serum treatment is immediately considered and instituted in appropriate cases. Should it be found that the patient's serum already contains agglutinating substances in high titre for the specific infecting organism, the use of antiserum is usually unnecessary.

Extreme care is exercised in carrying out tests of sensitization to serum in cases in which serum therapy is contemplated.

Technical details of sensitization tests and the method of serum administration are fully described in standard text-books and in the brochures of the manufacturers of sera in America.

All observers are agreed that in cases in which efficient concentrated serum is employed in early and adequate doses the results have proved eminently satisfactory and have produced a striking reduction in the death rate for pneumonia.

In a large series of cases analysed by Lord and Heffron[4] the expected mortality rate of Type I infections (25%) was reduced to 11·1% when adequate serum was applied early in the disease, that is, in the first ninety-six hours.

In cases of Type II infection an expected mortality rate of 41% was reduced to 27·2%.

From the practical point of view it appears to be a matter of extreme importance to find out the type incidence of pneumococcal infections in the community in Australia.

An exhaustive survey of the prevalent types has yet to be made; from the limited data at present available it is not improbable that infections of Types I and VII are responsible for a considerable number of cases. Should this be so, it is clearly our duty to apply serum therapy. Concentrated imported sera (Felton) are available commercially for Type I and II infections, although tariff impositions render the cost great and in some instances prohibitive. Up-to-date concentrated sera, comparable in efficiency to Felton's concentrated serum, have not been produced locally. Were adequate serum readily available, it is reasonable to suppose that the death rate from the pneumonic diseases in Australia could be materially reduced. The practising physician has, however, still to be educated up to the necessity for prompt examination of pneumonic sputa by a pathologist, so that the nature of the infective agent may be determined and adequate specific therapy instituted at an early stage of the disease. Until some such standards are reached our treatment of these diseases must be regarded as altogether inadequate as judged by world standards: a condition of affairs which it is hoped may soon be rectified.[1]

Treatment.

Ætiological Indications.

In dealing with infections by the pneumococcus, it is therefore obvious that apart from measures taken to prevent the spread of infection from patients the method of attack should be to invoke means of destroying the infective agent and employing measures to neutralize its effects upon the organs and tissues of the patient.

It has been shown that adequate serum therapy is at the present time the most effective therapeutic weapon.

Favourable reports have been received of the results of chemotherapy by the employment of sulphanilamide in infections of Type III pneumococcus, which bears some morphological resemblance to the streptococcus and for which no serum is at present available. Further advances in the field of chemotherapy are to be anticipated in the near future.

Pathological Indications.

Measures aimed at controlling the pathological processes in the lungs are limited in their application in dealing with this disease. Means may, however, be applied to counteract or diminish interference with respiratory function.

Adequate pulmonary ventilation is an obvious desideratum. The oxygenating capacity of the lung being diminished, measures to increase the supply of oxygen in the alveolar air are indicated. Apart from gross cyanosis, a rapid pulse rate (120 beats and upwards per minute), pronounced nervous symptoms and restlessness are regarded as indications for oxygen therapy.

The efficient administration of oxygen may be carried out in various ways.

1. The supercharged oxygen chamber may be used. This may be the method of election for patients severely cyanosed, with great respiratory

[1] In May, 1938, the National Health and Medical Research Council of Australia allotted a grant for the establishment of a pneumonia research centre at the Kanematsu Institute of the Sydney Hospital, where this work is now being carried on. Sputa may be sent in and rapidly typed, the results being communicated to the practitioner by telephone within a short space of time.

distress. The excessive cost and lack of facilities usually render this impossible in hospital services in this country.

2. Oxygen tents may be employed. Although readily applied, this method is restricted in usefulness by the rapid accumulation of carbon dioxide from expired air which occurs after a short period.

3. A mask with an expiratory valve may be used.

4. Post-nasal catheters may be used.

Apart from the use of an oxygen chamber, the employment of a mask with a flutter valve and the use of indwelling nasal catheters appear to be the most satisfactory and practical methods for the continuous administration of oxygen. By means of the mask it is claimed that a dose of six litres of oxygen per minute, controlled by a flow meter, is capable of enriching the alveolar oxygen to 35% or 42%; four litres per minute may enrich the alveolar oxygen to 32%.

The archaic methods of allowing the gas to bubble through water without measurement and delivering oxygen from a glass funnel dangled precariously over the patient's face should be no longer tolerated.

The tendency in the modern treatment of respiratory disease is to employ oxygen early and continuously rather than to delay its use until the patient has become cyanosed and his heart embarrassed, and pulmonary œdema is imminent.

The rôle of drugs in treatment tends to be progressively less. The consensus of opinion appears to be that digitalis and similar cardiac stimulants should not be employed as a routine measure; but should auricular fibrillation supervene, a rapid effect can be produced by the intravenous administration of "Digoxin".

Pituitrin or "Pitressin" appears to have a definite sphere of usefulness in cases in which abdominal distension is a feature. The routine use of adrenaline as a stimulant to counteract peripheral circulatory failures does not meet with universal acceptance overseas.

"Coramine" appears to have a sphere of usefulness as a potent respiratory stimulant in emergencies, but the hypodermic injection of strychnine and other drugs often employed is of very doubtful value.

Disturbance of patients by frequently unnecessary hypodermic puncture is not to be regarded as sound therapeutic procedure, and will surely be discarded so soon as modern specific methods of therapy are universally employed. Sulphanilamide has been employed in Type III infection. The use of nauseous mixtures—ammonium carbonate, potassium iodide and the like—is fortunately rapidly passing.

The use of a soothing linctus to allay excessive cough has justification on clinical grounds. The common expectorant mixtures containing potassium citrate *et cetera* should probably be restricted to cases in which pronounced bronchiolitis is present in association with bronchopneumonia. Alcohol as a stimulant and available energy producer apparently has warm advocates, particularly in cases in which nutrition is difficult or in which delirium and insomnia are prominent features. The routine use of stimulants of this type is less favoured than formerly. Where serum therapy and efficient means of oxygen administration are available, few drugs are likely to be employed except appropriate sedatives, of which morphine given hypodermically is probably the most useful, particularly when pain is a feature.

Symptomatic or Clinical Indications.

It will be universally conceded that meddlesome therapy is particularly contraindicated in a disease in which rest of body and mind is the primary indication. More is gained by competent nursing in good surroundings than by frequent movement of the patient to apply poultices and carry out local applications in support of which there is little evidence.

Pleural pain is, however, minimized by the application of heat by some heat-retaining substance of "Antiphlogistine" type, provided the patient is not disturbed too frequently and is allowed a maximum of rest without unnecessary interference.

Diet should be generous, and dehydration and loss of tissue turgor from depletion of chlorides is to be prevented. In cases in which there is evidence of dehydration and profound toxæmia, the employment of saline solution by intravenous injection is frequently beneficial unless adequate fluid can be taken by mouth.

Salt can be supplied by broths containing "Marmite", "Bovril" *et cetera* made with milk. The diet in each individual case is largely determined by the requirements of the individual rather than by fixed plan. It should be remembered always that the digestive processes may be inhibited by the disease, and that unsuitable food causing distention or abdominal discomfort is likely to be harmful.

Complications of Pneumococcal Infection of the Lungs.

Pulmonary Œdema.

Generalized pulmonary œdema is recognized as one of the most important complications of pneumonia in the acute stage, and is a potent factor in causing death, particularly in the pneumonic influenzal type of disease. Early recognition of its onset is of importance. The free use of oxygen is the most important therapeutic measure, aided by venesection in cases in which evidence of generalized cyanosis and congestion is pronounced in the absence of peripheral circulatory failure.

The use of morphine and atropine given hypodermically is frequently desirable to relieve respiratory distress and to ensure rest.

Pleural Effusion.

The occurrence of pleural effusion in the later phases of the disease should be carefully watched for and confirmed by exploratory puncture if any

doubt exists. Aspiration of serous fluid should be carried out, preferably by a closed method. Production of pneumothorax and increase in respiratory distress are thereby avoided.

Empyema.

Although several years have elapsed since the report of the Empyema Commission (Medical Research Council of the Privy Council) was published, the importance of recognizing the nature of the infective agent in purulent effusions is not yet invariably appreciated. In general, it may be stated that streptococcal infections should be dealt with by repeated aspiration, surgical drainage being delayed if possible until protective adhesions have formed to shut off the general pleural cavity. When the pneumococcus is the infective agent, fibrinous adhesions are formed earlier; and localized collections of thick pus, which cannot be satisfactorily dealt with by aspiration, may be dealt with in greater safety by surgical drainage at an earlier stage.

In all cases it is desirable to avoid collapse of the lung and the production of extensive pneumothorax, which is a condition of extreme danger. Recognition of these principles and the adoption of more efficient early treatment of the primary infection should tend to lessen greatly the mortality from complications of the disease.

Lung Abscess.

Lung abscess is an important, though fortunately a rare, complication of pneumonia, resulting from imperfect resorption of an inflammatory exudate and subsequent purulent softening. Its presence is to be suspected if fever is persistent or recrudescent after an apyrexial period, when no evidence of empyema is found. The diagnosis is usually confirmed by radiographic examination.

Treatment is beset with difficulty. At the annual general meeting of the British Medical Association at Belfast in 1937 the problem was exhaustively examined. The consensus of opinion expressed was that when the diagnosis of abscess was established, conservative methods were permissible for a short period (10 to 12 days) in the hope that the abscess might be drained naturally through a bronchus. It was claimed that this process might in some instances be facilitated by forcible manual percussion of the parieties over the affected area.

Bronchoscopic drainage might be reasonably attempted if drainage was imperfect, but radical surgical exploration by a two-stage operation should not be long delayed unless the condition of the patient rapidly ameliorated. Serial leucocyte counts may give some information as to the progress of the suppurative process. The results of surgical drainage are claimed to be reasonably satisfactory so long as interference is not unduly delayed.

Conclusion.

In conclusion, I would again stress the fact that the methods of treatment of pneumonia in common practice in our community are far from satisfactory; and if my efforts have been productive of a "divine discontent" in your minds and have inculcated an urge to strive for better results by the adoption of modern methods which have proved their worth abroad, this brief survey of the problem may not have been entirely devoid of useful purpose.

References.

(1) Leading article: "Pneumonia Mortality and Pneumococcus Typing Facilities", *The Journal of the American Medical Association*, Volume CIX, Number 23, December 4, 1937, page 1910.
(2) J. G. M. Bullowa: "The Management of the Pneumonias."
(3) F. T. Lord and R. Heffron: "Lobar Pneumonia and Serum Therapy", page 8.

TREATMENT OF PEPTIC ULCER BY A CONTINUOUS DRIP METHOD.

By M. M. O'Brien, M.B., B.S.,
Melbourne.

There is considerable experimental and clinical evidence that the acid factor is an important immediate or predisposing cause of peptic ulcer and of its persistence in a chronic form. Certainly in the treatment of peptic ulcer, control of gastric acidity is of the utmost importance. The great disadvantage of medical antacid treatment, apart from the danger of alkalosis, is its intermittency, especially its discontinuance during the long night period.

The ideal treatment would be one in which the gastric contents were kept neutralized throughout the whole twenty-four hours of the day. Such an ideal is attained in the continuous drip method of treatment. For this purpose an eminently suitable drug is a colloidal suspension of aluminium hydroxide, which has none of the several disadvantages of soluble alkalis in milk, as used by Winkelstein in America.

In a series of cases Woldman and Rowland, at Cleveland, Ohio, obtained very encouraging results. They demonstrated that in the vast majority of cases the ulcer may be completely healed in from seven to fourteen days. In only one patient in their series of twenty-three cases was an ulcer demonstrable after fourteen days' treatment; and that patient was considered to have had the ulcer for twenty years.

Clinical Histories.

The following are the clinical histories of two patients whom I have treated by the continuous drip method.

Case I.—M.G., a female, aged seventy years, was admitted to hospital on February 1, 1938. She gave the history that for the past six months she had had upper abdominal pain, coming on half an hour after meals. This pain was only slightly relieved by alkalis and would usually persist until she vomited. During the preceding four weeks the pain had been more or less continuously present. She had no appetite for food, and her bowels

were constipated. The patient was a thin, debilitated old lady; but, apart from epigastric tenderness, general examination revealed no abnormality. The test for occult blood in the fæces elicited a positive response.

A barium meal was given and an X ray examination made. This disclosed "a large penetrating ulcer on the lesser curvature of the stomach, associated with intense spasm, causing an hour-glass contraction of the stomach". Examination after a test meal revealed a high acid curve, the free hydrochloric acid rising to 52° and the total acid to 100° in two hours.

For seven weeks the patient received the ordinary medical treatment, including atropine and "Luminal". During this time there was no noticeable improvement clinically. Her general condition was unchanged; her appetite had not improved, pain was still present, especially at night, and vomiting was frequent.

It was then decided to institute the continuous drip treatment. This was continued for fourteen days with complete symptomatic relief. Her symptoms of pain, anorexia and vomiting had disappeared during the first few days of treatment.

X ray examination after a barium meal immediately after discontinuance of the treatment revealed "a small ulcer on the lesser curvature" and "some hyperperistalsis of the stomach".

Gastroscopy might have determined whether the small niche on the films was due to an ulcer or to a scar; but it was contraindicated by the patient's age.

The patient's general condition was very much improved and she remained free of symptoms on a number 2 ulcer diet, with small doses of colloidal aluminium hydroxide by mouth. She was discharged from hospital on May 1, 1938.

Case II.—J.J., a male, aged sixty years, was admitted to hospital with the history of several hæmatemeses during the past two days, two of which had been profuse. For the past six months he had had abdominal pain two to three hours after meals. This pain was relieved by food, and there had been periods of several days of complete remission of symptoms. His appetite had remained good, but he had noticed increasing weakness, dyspnœa on exertion, and some loss of weight. Apart from epigastric tenderness, general examination revealed no abnormality. The hæmoglobin value was 45%.

The patient was given hourly colloidal aluminium hydroxide by mouth and was allowed a milk diet. No further hæmatemesis occurred, and on the second day the continuous drip treatment was begun. A number 2 ulcer diet and some iron were given, and the patient improved rapidly. During the fourteen days of treatment he was free from symptoms and his hæmoglobin value increased by 15%. At the conclusion of treatment X ray examination after a barium meal revealed "no evidence of any ulceration or neoplastic change throughout the stomach or duodenum".

Gastroscopy disclosed a normal pylorus and a normal body of the stomach, with no evidence of anæmia, atrophy, ulceration or gastritis of the mucosa. The patient remained symptom-free and was discharged from hospital a fortnight later.

Discussion.

The result in Case I, we considered, was very convincing. Clinically, the patient had made no progress during seven weeks of medical antacid treatment, and then rapidly improved during the fourteen days of the continuous drip treatment.

The diagnosis in Case II must remain doubtful; but it illustrates the fact that hæmatemesis can be effectively treated by the continuous drip method — in fact, it is an excellent treatment. According to experimental work reported by Woldman, colloidal aluminium hydroxide will render gastric juice neutral to Töpfer's reagent, and in these circumstances the gastric hydrochloric acid becomes ineffective as an irritant or activator of pepsin. This allows a clot to form over the bleeding area, with subsequent organization and healing.

With regard to the technique of the treatment, the apparatus consists of a number 10 Levin tube, two bottles, a water reservoir and connexions. The Levin tube is passed down the nose to the lower end of the œsophagus. In our patients the indwelling tube caused very little discomfort; it was found that if the tube was passed through the cardiac orifice it would frequently become blocked. This tube is connected to bottle *C*, from which is siphoned the colloidal aluminium hydroxide solution. The rate of siphonage is controlled by the rate of flow of water from the reservoir *A* into bottle *B*. The preparation used was "Amphojel" (alumina gel), a 5% colloidal suspension of aluminium hydroxide. It was diluted with three volumes of water and allowed to run in at the rate of six to eight drops per minute. The rate of flow required can be determined by frequent testing of the reaction of gastric contents to Töpfer's reagent.

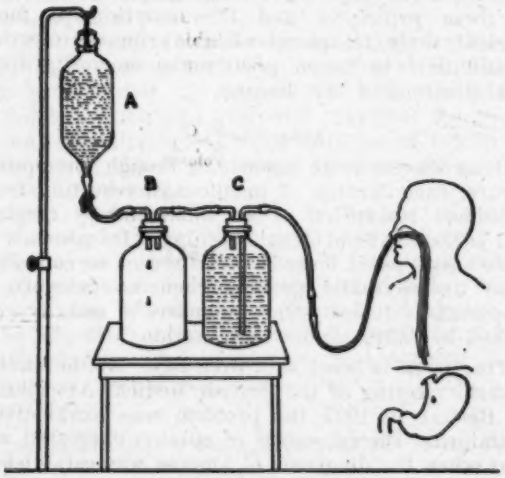


Figure I.

Apparatus used in the treatment of peptic ulcer by the continuous drip method.

The action and effects of colloidal aluminium hydroxide have been thoroughly investigated by Adams, Einsel and Myers, of Cleveland, Ohio.[1] They could find no evidence of any absorption of aluminium. No increase in the blood aluminium content and no increase of aluminium in the urine occurred. They demonstrated that in a patient under treatment there was a pronounced diminution in the amount of acid secreted. This does not indicate damage to the secretory cells; for even after prolonged treatment the response to injections of histamine is unchanged and the amount of acid secreted returns to normal after treatment is discontinued. With respect to the acid-base equilibrium of the blood, no significant changes were

demonstrated. The small changes noticed revealed a tendency for the total chloride content to rise and the carbon dioxide content to fall away from alkalosis. Lastly, in a large series of cases no idiosyncrasies to the drug, no vomiting, no toxic symptoms or complaints or evidence of gastric intolerance had been noted.

In conclusion, it appears that colloidal aluminium hydroxide is an excellent drug for the control of gastric acidity. One volume of a 5% solution will neutralize approximately twenty volumes of N/10 hydrochloric acid, and it has no ill effects other than slight constipation.

The continuous drip treatment with this drug represents a great advance in the treatment of peptic ulcer, as it is likely to succeed when other methods fail, and it will greatly reduce the time required for successful treatment.

Acknowledgements.

I am deeply indebted to Dr. Frank Niall, honorary physician to in-patients, Saint Vincent's Hospital, Melbourne, for his guidance in the treatment of the two patients and for his permission to present their clinical histories. In the treatment of the above two patients "Amphojel" was used. Supplies of this product were made available by a representative of John Wyeth and Brother, Incorporated.

Reference.

(a) W. L. Adams, I. H. Einsel and V. C. Myers: "Aluminium Hydroxide as Antacid in Peptic Ulcer", *American Journal of Digestive Diseases and Nutrition*, Volume III, April, 1936, page 112.

Reports of Cases.

PATHOLOGICAL REPORTS FROM THE CHILDREN'S HOSPITAL, MELBOURNE.

By Reginald Webster, M.D., D.Sc.,
Pathologist to the Children's Hospital, Melbourne.

XIII. Dermoid Cyst in the Cerebellum.

In view of the intimate relation which exists between the central nervous system and the ectoderm in embryonic life, it would not be matter for great surprise if intracranial dermoid cysts occurred much more frequently than they do.

The first step in the differentiation of the central nervous system is the formation of the medullary plate, a thickening of a strip of ectoderm along the axial line. This is followed by a depression in the middle portion of the medullary plate, which defines the medullary groove. The elevated edges of the medullary groove approach each other and fuse, and thus is formed the medullary tube, the anterior portion of which enlarges and becomes subdivided into the three primary vesicles of the brain.

It might be anticipated that the ensuing complexities involved in the development of the brain and its ventricles, the formation and union of the cranial bones, would provide many opportunities for the fortuitous detachment and sequestration of those dermal rests, to which, by common consent, pathological dermoid cysts are referred. Possibly epiblastic inclusions frequently occur, but are seldom permitted to attain any further development. Certain it is that dermoid cysts within the cranium come within the category of pathological curiosities. I. Douglas Miller,(a) of Sydney, in reporting the occurrence of a dermoid cyst of the cerebellum in a girl, aged eleven years, in 1937, drew attention to the fact that Cushing, in his unparalleled experience of intracranial tumours, recorded only three dermoid cysts, and mentioned that Dandy had met with one only.

In these circumstances, the example from the pathological museum of the Children's Hospital, Melbourne, photographed to provide Figure XX, is worthy of record. This particular dermoid cyst arose within the skull of a little girl, aged seven years, followed tradition in adhering to the middle line, and exemplified the predilection of such cysts for the region of the internal occipital protuberance or inion.

Unfortunately the clinical record of the patient from whom this specimen was obtained cannot be traced. It may be taken, however, that there was little doubt attending the diagnosis of cerebellar tumour; for the operation of suboccipital decompression, which the child survived for a few hours only, was performed on November 18, 1927. Although a summary of clinical aspects would have been desirable, it is not essential to my present purpose, which is to present and discuss a rare specimen.

At the autopsy, beyond noting that a large tumour bulged posteriorly between the lobes of the cerebellum, I did no more than remove the brain and place it in a specimen jar containing a 10% solution of formalin. On this, as on many similar occasions before and since, I was obliged to resist the demands of impatient onlookers that the brain be immediately dissected. To do so before the brain was hardened would have been to incur great risk of the ruin of the specimen as a permanent preparation.

A sagittal section through the brain in the middle line, made a week later, disclosed a globular "tumour", situated centrally between the lobes of the cerebellum. A glance at the illustration (Figure XX) shows the vermis of the cerebellum, flattened and atrophied as the result of compression between the tumour and the unyielding *tentorium cerebelli*, riding on the dorsal surface of the intruding mass. A little teasing of the cut surface of what still appeared to be a tumour disclosed the presence of hairs; what was at first regarded as tumour substance was then found to be a greasy smegma, and the true nature of the specimen became obvious. From one half of the cyst, that in contact with the right lateral lobe of the cerebellum (Figure XXI), the sebaceous contents were evacuated, and the cyst wall, very thin and resembling nothing so much as a piece of hair-bearing parchment, was lifted out of the bed which the dermoid had made for itself in the lateral lobe of the cerebellum. The cerebellar tissue was compressed, but otherwise not damaged; and on the medial aspect of the right lateral lobe was a concavity in which a golf ball might have been placed (Figure XXI).

Microscopic sections of the cyst wall provided all necessary confirmatory evidence that the cyst was of dermoid nature: compressed stratified squamous epithelium, sebaceous glands, and hair follicles in abundance.

The left half of the brain was prepared for museum mounting, steps being taken to ensure that the lateral ventricle was maintained in the state of dilatation in which it was found (Figure XX). This precaution was not taken with the right half, as it was not intended to mount it as a permanent preparation (Figure XXI). Consequently the right lateral ventricle appears collapsed; but at the first examination of the brain both lateral ventricles were symmetrically dilated in the manner shown in Figure XX.

The globular-shaped dermoid cyst measured 4·5 centimetres in diameter, and a further reference to the illustration will lead to an appreciation of the manner in which it pressed down on the roof of the fourth ventricle, and by the exertion of forward pressure also produced an extraordinary angulation of the brain stem. Such distortion of the brain stem was alone sufficient to bring about an obstruction to the flow of cerebro-spinal fluid at the most vulnerable point in the ventricular system, the aqueduct of Sylvius. It would seem that a degree of

angulation of the brain stem such as is shown in Figure XX could not fail to result in occlusion of the Sylvian narrows. The closure of the foramen of Magendie and the foramina of Luschka effected by the presence of the dermoid cyst is also apparent from the illustration. If pressure on the great vein of Galen is admitted as a contributing factor in the production of the hydrocephalus which led to the greatly dilated lateral ventricle and widely open foramen of Monro shown in Figure XX, this cyst is strategically situated to nip the great vein at the point where it curves round the splenium of the *corpus callosum* in its passage to the anterior extremity of the straight sinus.

In describing this specimen as a dermoid cyst in the cerebellum, I was careful in the choice of the preposition, for it seemed that the cyst, though "in" the cerebellum, was not "of" it. But is it really "in" the cerebellum? The vermis and lateral lobes are displaced and subjected to pressure; but the cyst is really outside the cerebellum and does not invade it at any point. I submit, therefore, that the cyst, to be designated accurately, should be presented as a dural dermoid cyst, originating in the *tentorium cerebelli* or the *falx cerebelli*.

Bland Sutton,[2] whose descriptive term "sequestration dermoid" has maintained its place in orthodox text-books for many years, emphasizes that dermoids of the scalp and *dura mater* are commonly situated over the anterior fontanelle and the occipital protuberance. Dermoids in the region of the occipital protuberance (inion) may lie on the inner aspect of the occipital bone and are nearly always in relation to the *tentorium cerebelli*.

Early in embryonic life the *dura mater*, which represents the primitive cranium, is in contact with the skin, a relationship which is preserved along the various sutures even for a year or more after birth, and persists longest in the region of the bregma and in the vicinity of the inion. As the *tentorium cerebelli* arises in the first instance as an infolding or crease in the *dura mater*, it follows that the opposed surfaces of the tentorial lamellæ have originally been in contact with the skin. As the posterior margins of the recess formed by the crease in the *dura mater* come together, a portion of the skin may well become detached and imprisoned between the layers of the tentorium. Should such a sequestered piece of dermis retain its vitality it may ultimately give rise to an intracranial (dural) dermoid.

Ewing[3] describes dural dermoids as lying close to the skull, growing deeply and pushing the pia before them. Arising relatively late in embryonic life, after the skin is formed, they usually lie in the median line in the *tentorium cerebelli*, at the internal occipital protuberance or *torcular Herophili*, or they may be epicranial.

The position and relations of the particular dermoid cyst under discussion, as I have endeavoured to describe them, indicate that strictly regarded it is neither "in" nor "of" the cerebellum, but that it is of dural origin, and has merely pushed the cerebellum before it as it grew from an epidermal inclusion between the two layers of the *tentorium cerebelli* or possibly those of the *falx cerebelli*.

From the description given by I. Douglas Miller[1] of the conditions he encountered at operation upon the child whose lesion he reported as a dermoid cyst of the cerebellum, I would venture to suggest that in that instance also the dermoid was essentially dural in origin.

The nature of the specimen I have presented was never in doubt. The presence of hair and the characteristic microscopic features in a section of the wall of the cyst established its identity beyond question, and it was deemed not necessary to pay any further attention to the greasy contents. Consequently, the presence of cholesterin, in all probability a prominent constitutent of the smegma, was not demonstrated.

Mention of cholesterin raises the vexed question of cholesteatoma. Ewing[3] describes a cholesteatoma as a tumour composed of lamellated waxy or scaly material, enclosed in a wall of stratified squamous cells, and as occurring in all parts of the brain, ventricles and basal regions. It usually lies near the middle line and is regularly connected with the meninges. He quotes Boestrom as authority for the statement that all cholesteatomata arise from embryonic epidermal inclusions.

There is a strong suggestion in this description of the *tumeurs perlées* of Cruveilhier, in discussing which Percival Bailey,[4] noted co-worker with Harvey Cushing, writes impatiently of the confusion which has been created by designating true pearly tumours as cholesteatomata. Bailey suggests that the term cholesteatoma should be restricted to those collections which arise in the middle ear in connexion with chronic otitis and extend into the cranial cavity. That such should be identified with true pearly tumours, of startling resemblance to mother-of-pearl both in tint and lustre, seems to Bailey intolerable. He does not express himself thus emphatically, but this is the impression gained from reading between the lines.

A true pearly tumour has never come within my observation, or, if it has, I have not recognized it. There is an undoubted fascination about Bailey's description of the *tumeurs perlées*, with their smooth silky surface, from which, with a knife blade, flakes which show beautifully the characteristic lustre may easily be separated. Again, a true pearly tumour in a setting of reddish brain tissue makes a "beautiful picture", and its beauty seems to be enhanced by its fragility, for it "crumbles like a puff ball when touched".

Bailey writes fondly and with æsthetic appreciation of the pearly tumours, and, as has been indicated, protests against their inclusion among cholesteatomata merely because cholesterin crystals are to be demonstrated in their contents.

Pearly tumours are rarely seen in man, only sixty-two genuine examples having been reported, but occur frequently in the lateral ventricle of the horse. They are meningeal growths and not brain tumours proper, and lie always beneath the arachnoid, in the pia. Controversy has centred round their origin, whether epithelial or endothelial, with the weight of evidence apparently in favour of an epithelial origin. The only feature they have in common with dermoids is that they contain cholesterin crystals; they never contain hair, and confusion should never arise between *tumeurs perlées* and dermoids. The two are so dissimilar that it is doubtful if pearly tumours have a natural place in a discussion on intracranial dermoids. Still, *tumeurs perlées* have been considered to arise from embryonic epithelial cell rests, and apart from the question of their origin, have a peculiar attraction all their own.

References.

(1) I. Douglas Miller: "Dermoid Cyst of the Cerebellum", *The Australian and New Zealand Journal of Surgery*, Volume VII, Number I, July, 1937, page 74.
(2) J. Bland Sutton: "Tumours Innocent and Malignant", Seventh Edition, London, 1922.
(3) Ewing: "Neoplastic Diseases."
(4) P. Bailey: "Cruveilhier's 'Tumeurs Perlées'", *Surgery, Gynecology and Obstetrics*, Volume XXXI, 1920, page 390.

A CASE OF UNILATERAL FUSED KIDNEY (CROSSED ECTOPIA).

By Oswald J. Ellis, M.B., Ch.M. (Sydney), F.R.C.S. (Edinburgh), F.R.A.C.S.,

Honorary Medical Officer, Mater Misericordiæ Hospital, Newcastle, New South Wales.

Although a considerable number of cases of unilateral fused kidney have now been reported, it would still appear to be one of the rarest types of congenital abnormality of the kidney. I have therefore considered it worth while to record the following case.

The patient, Miss B.A., was aged ten years. The mother, who has been a nurse, complained that the child had suffered from a persistent urinary infection with *Bacillus*

coli communis for four years, manifested by periodically recurring attacks of fever, accompanied by urinary frequency and irritation. Apart from these attacks the child had no symptoms whatever. During this time she had received treatment intermittently; but no attempt had been made to carry out a full urological investigation.

On examination the patient was observed to be a well developed, apparently healthy child. Nothing abnormal was palpated on abdominal examination. The external genitalia were normal.

Dr. Ethel Byrne's examination of a catheter specimen of urine revealed occasional pus cells, and no *Bacillus coli communis* in a microscopic film. However, *Bacillus coli communis* was revealed by culture.

A "Uroselectan" radiogram was then carried out (Figure I) and proved extremely interesting. This shows that the right kidney is absent from its normal position, and situated to the left of the mid-line below the left kidney. The left kidney is seen to be normally placed, but with its pelvis rotated somewhat forwards. The ureter of the left kidney does not pursue a normal course, but is bowed outwards and can be followed to the region of the left sacro-iliac joint. This becomes of significance later. There are two other areas of opacity visible: the smaller one situated to the left of the interspace between the fourth and fifth lumbar vertebræ, and the larger situated anteriorly to the abnormal sixth lumbar vertebra. The exact interpretation of these two shadows was doubtful, and it could not be definitely decided whether the upper shadow represented the pelvis of the lower kidney and the larger shadow represented some dilatation in the course of the right ureter, or whether the larger shadow represented the pelvis of the lower kidney. The smaller shadow would then represent a dilated upper calyx of the same kidney.

In a further endeavour to clear up this doubtful point, a cystoscopic examination, preceded two hours previously by the administration of fifteen grammes of urea, was carried out under gas anæsthesia. The bladder was normal and both ureteral orifices were seen to be normally placed. Catheters were passed up both ureters, but could not be advanced further than eleven centimetres on either side. The patient was then allowed to come out of the anæsthetic. A preliminary screening showed both catheters in position in the corresponding ureters. The kidney pelves were then injected with a 12·5% solution of sodium iodide, and pyelograms were obtained. Unfortunately the child started to cry and become restive immediately the injection was commenced on either side, with the result that the best definition has not been obtained in the skiagrams. However, they are clear enough to show the condition present.

In the pyelogram of the upper kidney (Figure II) the previous results are confirmed; but it is also seen that there is an acute kink in the ureter at the level of the left sacro-iliac joint. This represents the point beyond which the ureteral catheter would not pass.

In the pyelogram of the lower kidney (Figure III) it would now appear that the shadow anterior to the spine represents the dilated pelvis of the lower kidney, while the smaller shadow higher up is merely a dilated calyx. Evidently there is some obstruction to the right ureter. In this skiagram also, and to a lesser extent in the others, the kidney shadows appear to be fused.

Examination of the catheter specimens obtained from each kidney showed they were both sterile, attempts at culture being unsuccessful. The figures of the urea concentration test were as follows:

Before urea	3·8%
One hour after	3·0%
Two hours after	3·0%
Three hours after (upper kidney) ..	4·6%
Three hours after (lower kidney) ..	4·2%

From a study of the pyelograms it was thus apparent that we were dealing with the rare condition of unilateral fused kidney or crossed ectopia.

The only other congenital abnormality discovered in this child was the presence of six lumbar vertebræ. The child is well developed for her age, bright and intelligent, and apparently quite healthy. In these circumstances, and in view of the negative bacteriological findings, it was doubtful whether a laparotomy, with a view to removing possible ureteric obstruction, would accomplish any useful result. In any case, the parents were averse to this, and so the diagnosis has to rest at this point. I propose to keep her under periodical observation with a view to possible future developments.[1]

Comment.

This case is very interesting and would point the moral that all cases of persistent urinary infection are worth a full urological investigation.

Acknowledgement.

I am indebted to Dr. Ethel Byrne for the pathological investigations, and to Dr. A. B. Sullivan for the X ray films.

Reviews.

SEXUAL DISORDERS.

Dr. Max Huhner, author of "The Diagnosis and Treatment of Sexual Disorders in the Male and Female",[2] is a distinguished member of the medical profession in New York. Besides being a urinary surgeon at Bellevue Hospital and an assistant gynæcologist at Mt. Sinai Dispensary, he is a fellow of several medical associations. His qualifications to write upon the subject are therefore undoubted and he has produced a work of great merit.

The book is divided into four parts. Part I treats of sterility; Part 2 is devoted to a wide survey of impotence; Part III consists of three chapters on masturbation; while Part IV contains thirteen chapters on the various types of sexual disorders. The whole range of sexual abnormalities in both male and female is thus dealt with very thoroughly. The most important chapters in the book, however, are those treating of masturbation and withdrawal (*coitus interruptus*).

Dr. Huhner holds very strong views on the causes and treatment of chronic masturbation. Briefly his belief is that once the practice of masturbation has commenced, there gradually results a chronic congestion of the posterior urethra which is, of course, normally congested during sexual excitement. This chronic congestion causes a constant stream of nervous impulses of an erotic nature to pour into the nervous system. Then ensues a desire for relief of tension and the sexual act is again performed either normally or by masturbation. Roughly this is the mechanism in both sexes. Dr. Huhner regards the female urethra as one of the most highly erotogenized zones in the female.

The author's treatment, therefore, is directed towards a reduction of this congestion by means of injections of zinc sulphate with special syringes. He is positive that so far as the male is concerned this is the only sure treatment. He is not so sanguine of its efficacy in the female, for he asserts that it is useless for the male to endeavour to cure himself by an exercise of will and yet counsels the female to attempt it. "We must try", he writes, "to develop her will power and self control." In contradistinction he states regarding the male masturbator: "It is useless to talk to a confirmed masturbator of self-control because he cannot control." It is difficult to reconcile these contradictory statements. It is not true that the chronic masturbator cannot exercise will power and control. Under proper guidance some do, and cure can and does result.

These chapters on masturbation contain much wisdom, but they are rather weakened by a misconception of what the author terms the Freudians' ideas on this subject. He states, and states it with wearisome reiteration, that the

[1] Since this report was written the patient has left the Newcastle district.

[2] "The Diagnosis and Treatment of Sexual Disorders in the Male and Female, including Sterility and Impotence", by M. Huhner, M.D.; 1937. Philadelphia: F. A. Davis Company. Demy 8vo, pp. 503. Price: $5.00 net..

Freudians advise masturbation. He quotes as his authority extracts from a work (title not given) by Dr. William Stekel which was published in 1923. Among these extracts are the following: "All the bad effects attributed to masturbation exist only in the imagination of the doctor", "There are some people who are afraid of contracting venereal disease and others who for ethical reasons abstain from coitus before marriage and it is for both these classes that masturbation comes in as a great help to preserve their continence". Further on the author declares that "the psychologist does not believe in a cure for masturbation" and quotes the alleged Freudian dictum: "There are many people who could not live without masturbation."

We cannot say whether Dr. William Stekel actually holds these opinions or once expressed these views in the book (unnamed) from which Dr. Huhner quotes; but the very citation of these statements as the opinions held by Freudians and psychoanalysts only shows how very ignorant the author is of the ideas on masturbation held by Professor Freud and his followers. Dr. William Stekel cannot be classed as a Freudian; he parted company with Professor Freud many years ago and all psychoanalysts would reject him as in any way a representative of their views. It is apparent that Dr. Huhner has a strong bias against the Freudian school and it is a major fault in a book that has few defects that he allows himself to display prejudice by his repetition of these erroneous views.

Dr. Huhner could have shortened his book a good deal by omitting the many repetitions on different aspects of his subject. Another defect of the work is the absence of any reference to the use of male sex hormones, either natural or synthetic (terasterome bodies), in conditions such as impotence, or to the use of anterior pituitary extracts in impotence, aspermia *et cetera*. We are not quite certain when the book was actually written or rewritten, for the preface is undated. However, as the date of the publication is 1937, there should have been shown some knowledge of the researches and clinical records of the sex hormones.

The work is completed by a bibliography of over 600 references and an excellent index.

ESSAYS IN BIOCHEMISTRY.

ON the occasion of the seventy-fifth birthday of Sir Frederick Gowland Hopkins, professor of biochemistry in the University of Cambridge, a volume entitled "Perspectives in Biochemistry" was presented to him.[1] It comprises thirty-one essays by past and present members of his laboratory. The essays cover a wide range of subjects, including among others physiology, bacteriology, nutrition, medicine and genetics.

In the preface the editors state that the aim of the writers has been to indicate the most promising lines of advance in the various fields which they survey, and while maintaining a due standard of criticism, to speculate a little on the likely paths of future thought and discovery. The essayists include such well-known names as J. B. S. Haldane, R. A. Peters, A. von Zent Gyorgyi, J. Murray Luck, Edward Mellanby and other brilliant experimentalists.

The whole volume is offered as a token of deep gratitude to the doyen of English biochemistry, for, as one contributor writes, "with the growth of biochemistry to its present status the name of Hopkins will always be associated". The subjects of the essays include interesting themes, notable among which are the lack of meaning of the terms "life" and "living", the economy of the bacterial cell, biochemical approaches to the study of the functions of the nervous system, drugs and mankind, and the social implications of biochemistry. A splendid description is given of the evidence which indicates that vitamin *D* is now to be regarded as a mixture of closely related chemical compounds. In another contribution attention is drawn to the increase in drug addiction since the Great War. Particularly does this apply to the use of morphine, heroin and cocaine. It is pointed out that in Egypt in 1930 the number of drug addicts was 500,000, or 3% of the population.

The book throws much light on various aspects of biochemistry, the study of which, as the concluding essayist remarks, increases man's control of his destiny.

[1] "Perspectives in Biochemistry: Thirty-One Essays Presented to Sir Frederick Gowland Hopkins by Past and Present Members of His Laboratory", edited by J. Needham and D. E. Green; 1937. London: The Cambridge University Press; Australia: S. Jaboor. Demy 8vo, pp. 369. Price: 15s. net.

ELECTROCARDIOGRAPHY.

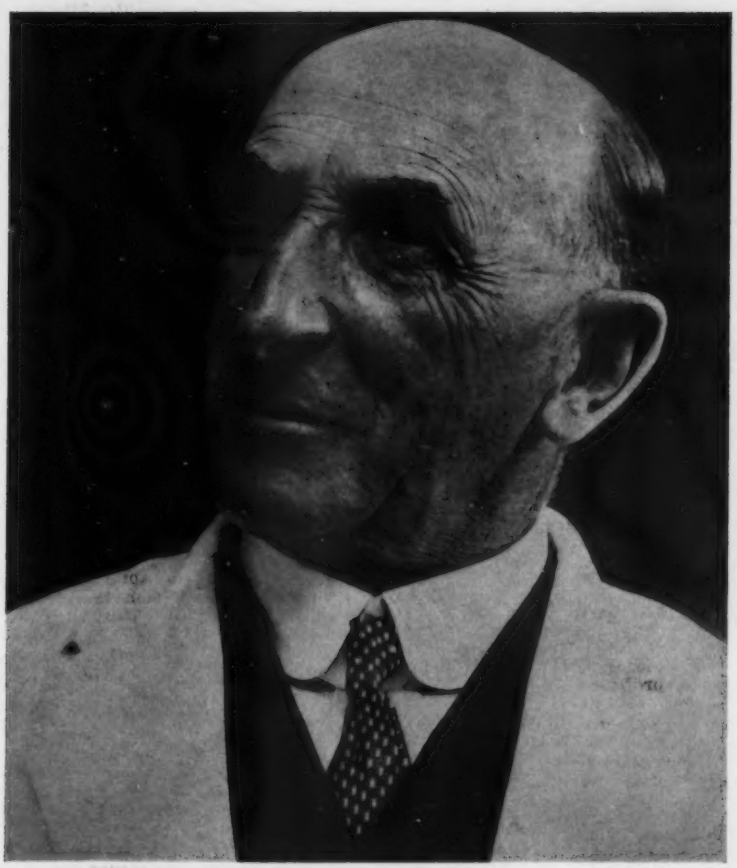
"THERE is need for a treatise on electrocardiography for use by the general practitioner, the medical student, and the specialist exclusive of the cardiologist, that they may learn the value of the electrocardiogram in the diagnosis of cardiac disease and learn to correlate the electrocardiographic findings with their clinical data. To meet that need this volume is written." Chauncey Maher writes thus in the preface to the second edition of his book "Electrocardiography".[1] The volume is a small one, printed in readable type and profusely illustrated with cardiograms. We believe these were taken direct from actual tracings. Their plain, semi-diagrammatic reproduction is a welcome relief from the often poorly defined photographs in similar books. The text is concise and reasonably easy to follow. The cardiograms are interspaced with the appropriate text and not inserted at the end of the chapter or book. In fact, the whole volume can be read in two or three sittings, as illustrations comprise almost two-thirds of its pages. Whilst in this respect it could really be termed an atlas, it is a book for complete study rather than for reference.

The early pages are devoted to a description of the physics and mechanics of electrocardiographic technique. Much mathematical theory has happily been omitted, and yet enough of the fundamental principles has been retained to permit the newcomer to the subject to understand the basis of interpretation.

In the opening chapter the author briefly reviews the cardiological field from the clinical viewpoint, using the criteria for the classification of heart disease which were adopted originally by the New York Heart Association and subsequently by many others. The basis of this scheme is that no cardiological diagnosis is complete without a statement of the ætiology, anatomy and abnormal physiology of the lesion made according to certain clearly defined standards. Above each cardiogram reproduced in the book is a description of the findings. Great care has been taken by the author to correlate clinical and cardiographic evidence. Indeed, in spite of a warning statement in the last chapter, it is possible that the beginner may obtain an exaggerated impression of the value of the machine. Dr. Maher apparently favours the theory of multiple ectopic foci rather than that of circus movement as an explanation of auricular flutter and fibrillation, although here, as elsewhere, he wisely refrains from dogmatism. He is impressed with the greater seriousness of right than left bundle branch block (using Wilson's terminology). He believes that high degrees of left axis deviation are not necessary to produce *T* wave inversion in the first lead. It would seem that hypertrophy or myocardial damage is required to bring this change about. He speaks of "vagal pressure" in the neck, when surely it is now agreed that it is carotid sinus pressure which may sometimes be beneficial in tachycardia.

"Brevity and conciseness have been sought for. Controversial literature and extensive bibliography may be readily found elsewhere", he says. The latter have no place in a manual for students, to whom this little volume can be particularly recommended. Its illustrations alone make an easy study for the beginner and good practice for the erudite.

[1] "Electrocardiography", by C. C. Maher, B.S., M.D.; Second Edition; 1937. London: Baillière, Tindall and Cox. Super royal 8vo, pp. 270, with illustrations. Price: 18s. net.



Colin Mackenzie

The Medical Journal of Australia

SATURDAY, OCTOBER 1, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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TUBERCULOSIS IN CHILDREN.

Happily, tuberculous disease in Australia does not kill or cripple so many infants and young children as it does in some other countries. Nevertheless bone and joint tuberculosis is sufficiently common in this continent to demand special facilities for the prolonged treatment of affected children in the larger centres, and tuberculous meningitis is not rare among the very young. Yet these manifestations of the activities of the tubercle bacillus are not the most important aspects of the problem, for it is essential to recognize childhood as the time when the destiny of the man to be is shaped as regards his chance of contracting pulmonary tuberculosis in the future. It is illuminating to look at the figures from Europe. The statistics of Armand de Lille, quoted by Alvin E. Siegel in an estimate of the modern position of tuberculosis in childhood, reveal that 60% of the infants and children in contact with tuberculous parents present characteristic evidence of infection before the death of the parent, and of these, two-thirds die from acute forms of the disease and only one-third have chronic lesions.[1] The exact percentage applicable to the various Australian urban and rural populations matters little at the moment. The point is that quite a large percentage of children who are in contact with sufferers from tuberculosis are demonstrably infected. Clinically sound they appear to be, and in fact the experience of tuberculosis clinics is that little is to be found on examining these children with a stethoscope. More useful information is afforded by a simple study of their general nutrition. A skin test for sensitivity reveals that infection has occurred, and X ray examination may reveal shadows in the hilar regions of the lungs, suggesting foci of infection, perhaps healed or quiescent, but a potential danger. Myers and other authorities quoted in the same article refer to the parenchymal shadows, seen in skiagrams of the lungs of children, the nature of which is often in doubt, and they lay down the principle that if such a shadow persists longer than a month in the skiagram of a child sensitive to tuberculin it is usually tuberculous in origin. Schick believes that the primary tuberculous complex in children has a limiting effect on any superimposed infection which may occur at a later date. Others, however, feel less sure and hold that the primary infection may be an asset or a handicap, according to circumstances. Certainly there has been a great deal of confusion about the subject of sensitivity or allergy: it has too often been regarded as *per se* an evidence of immunity. As Armand de Lille says, the adult patient is allergic to tuberculin, and this fact tends to produce localization, especially in the lungs, in which there are so often primary foci and which act as a kind of screen for the entry of tubercle bacilli into the blood stream.

As regards prophylaxis, Siegel says that there are several paths to be followed. These are the recognition of active disease in adults and the segregation of these patients as far as possible; the search for evidence of childhood infection by skin tests and X ray examination; the surveillance of children known or suspected to be infected; and, lastly, the use of Bacille-Calmette-Guérin vaccine. The experience of a number of workers seems to guarantee the

[1] *The American Journal of the Medical Sciences*, January, 1938.

harmlessness of this latter method, though the oral use of the vaccine seems to be much less effective than its administration by injection.

One of the most encouraging studies is that of P. W. Beaver, who recorded the results of investigation of 4,982 children during the years from 1926 to 1934. He came to the conclusion that tuberculosis in children was decreasing rapidly, and he actually believes that it should become a rare disease if this rate of decline is maintained. But it must be remembered that in Australia manifest tuberculosis in children is not a serious problem as a clinical disease; yet pulmonary tuberculosis is sufficiently common to warrant constant watchfulness over all adolescents. The general decline of the incidence and mortality of tuberculosis must not mislead us. It has rightly been regarded as a comfortable doctrine which tends to hinder public health activities, as was pointed out during the congress in Adelaide last year. The importance of the exposure and infection of children cannot be overemphasized. It is this generation of children, infected through ignorance and carelessness, that may provide us with the next crop of adult sufferers from pulmonary tuberculosis. Arvid Wallgren, writing of the relation of the childhood infection to the adult disease, points out that we may expect a pulmonary tuberculous condition irrespective of the site of the primary focus.[1] No child with foci of infection is really safe, and Wallgren draws attention to the risk when the second danger period is reached. This period begins at puberty and lasts throughout two or three decades, during which pulmonary disease may appear. The fate of an individual depends considerably upon the degree of resistance he possesses, and this will in turn depend upon various factors in environment and in the body of the person himself. It is not necessary to become involved in the many controversial questions in the study of tuberculosis. We are on sure ground in all the important aspects of prevention, and it must be to the advantage of the community to lay stress repeatedly on the attention which must be directed to the child and the adolescent.

[1] *The Lancet*, February 19, 1938.

Current Comment.

THE CAUSES OF NEONATAL DEATHS.

Modern study of the causes of infant mortality has been followed by a decrease in the death rate of infants and young children. There has been no decrease in the stillbirth rate, nor in the death rate of infants under two weeks of age. We are still ignorant of the precise nature of many conditions which cause the death of the fœtus during labour and of the newly born child. Large general hospitals usually have well-equipped autopsy rooms and pathology departments, in which the deaths of adults may be investigated with completeness. Similar time and facilities have not often been devoted to examination of the bodies of stillborn infants and those who have died shortly after birth. Yet, as Edith Potter observes, it is often much more difficult to determine the cause of death of an infant than of an adult.[1] The autopsy on the fœtus or newly born infant is quite different from the autopsy on a mature person; for the most important causes of death in the neonatal period are those which prevent the normal establishment of extrauterine existence, and which consequently never function in later life as a cause of death. There is a small group of infants who fail to live because of congenital abnormalities, and a small group who die from infections. The deaths of the remainder are largely due to *ante partum* and *intra partum* obstetric complications. Birth injury is, of course, a well-recognized cause of neonatal death. The most common form is intracranial injury; but abnormal manipulation of the neck may give rise to injury of the spinal cord, or undue compression of the fœtal abdominal wall may lead to intraperitoneal hæmorrhage from rupture of the liver or the adrenals. Massive hæmorrhages into the substance of the suprarenal gland may distend its capsule enormously, so that a gland which under normal conditions would weigh three to four grammes may weigh 35 grammes or more. In Potter's opinion the administration of drugs and anæsthetics during labour probably plays a part in the causation of stillbirth; but this is difficult to assess.

The macroscopic and microscopic appearances of the tissues of the newly born are well described by Potter, and the variety of the information included by her in this study bespeaks a wide experience. It is often difficult to determine the cause of death in the newly born; if the body of the infant is the only material available, it is frequently impossible. An adequate clinical history is necessary, the placenta and cord must be examined, and microscopic examination of the viscera may be essential. Of how little value, then, are statistics based on death certificates of cases in which no investigation has been held!

[1] *Archives of Pathology*, May, 1938.

The practical vigour of American investigators has shown that this work is not only of great interest, but of great value. Bundesen, Fishbein, Dahms, Potter and Volke found that a large majority of death certificates of infants in Chicago did not name the true cause of death.[1] In many cases it was not known and could not be established without investigation. During 1936 a campaign had been undertaken to increase the number of satisfactory necropsies on infants. The result was that whereas only 35% of the deaths of infants occurring during 1936 were properly investigated, satisfactory necropsies were performed on 53% of infants who died during 1937, and on 83·5% of infants who died during the first half of 1938.

These authors present an analysis of the causes of the neonatal deaths investigated; their analysis of the causes of stillbirth is not yet published. The data thus collected are well worth a careful study. The summary may be quoted here: "A large number of factors contributing to deaths of infants under two weeks, and particularly those under one day of age, may be controlled by the knowledge now available. Improvement in obstetric care, both *ante partum* and *post partum*, better hospital nursery care, the avoidance during delivery of dangerous procedures, and of the injudicious use of drugs (which figures show to be definitely hazardous), the use of measures to prolong pregnancy to term, such as 'Progestin' and vitamin *E*, and the employment of proper methods of resuscitation, should bring a definite reduction in neonatal mortality."

TREATMENT OF A PARATYPHOID CARRIER WITH SULPHANILAMIDE.

Undoubtedly there is a tendency to vaunt sulphanilamide as the elixir of life or at least as the universal remedy. While the prudent man should accept enthusiastic first reports with caution, he should nevertheless be progressive and show willingness to try this powerful agent in many types of infection. A. T. Bazin, of Montreal, reports the single case of a carrier of paratyphoid *B* organisms in which this drug was used with apparently brilliant success.[2] He quotes an estimate that 3% to 4% of sufferers from typhoid fever become permanent carriers of the infection and points out that the carriage may be "open", when the organisms are present in the fæces or urine or both, or "closed", when they are present only in bone abscesses. The gall-bladder is well known as the focus from which the bacilli proceed to the bowel of the carrier, and the existence of gall-stones or their subsequent formation appears to make it more likely that the infection will be permanent. Typhoid bacilluria does not constitute a serious problem in the carrier, as it disappears within a few months at the most; the real problem is sterilization of the biliary passages. Cholecystectomy has been performed on a number of patients whose gall-bladders have been found to be infected, but this measure is only likely to be successful when the gall-bladder alone is at fault and when the biliary passages in the liver are not the chief source of infection.

Bazin's patient was a woman, aged fifty-five years, who was employed as a nursemaid. When a child under her care fell ill with paratyphoid fever, the stools of all contacts were examined, and in this way the woman was proved to be a carrier. Nothing relevant in her history was discovered, except the fact that a member of a family with whom she had lived previously had suffered from an illness diagnosed as paratyphoid *A* fever. Repeated cultures of the fæces of the nursemaid revealed growths of *Bacillus paratyphosus B*, and from a specimen of bile obtained by means of a duodenal tube a pure culture of this organism was grown. When a Graham test was made, the gall-bladder could not be seen, but shadows suggestive of stones were observed. Operation was performed and a single stone was found in the cystic duct, the gall-bladder being thickened and opaque. The common bile-duct also contained stones; these were removed and the gall-bladder was excised. Bile drained freely from a tube inserted into the common duct, and from this the paratyphoid organism was again cultured. At this stage the intern who had been doing the surgical dressings contracted paratyphoid fever. The drainage tube was therefore removed to minimize the danger to others. Hexamine was given to the woman without result, but following the administration of sulphanilamide, attempted culture of the organisms from the stools was unsuccessful. The dosage of the drug was increased to 6·0 grammes (90 grains) a day, and two separate courses were given, the first of a week's and the second of ten days' duration. The patient complained of headache and dizziness, but careful blood examinations showed no evidence of sulphæmoglobinæmia and no reduction of red or white cells. During the next month repeated attempts at culture from the stool and even the duodenal bile showed no traces of *Bacillus paratyphosus*.

In his comments Bazin distinguishes the carriers whose liver bile is infected from those in whom the gall-bladder alone is involved. He mentions a third group in whom the intestine is infected without an associated infection of the biliary tract. Discussing treatment with sulphanilamide, he wisely remarks that one swallow does not make a summer. Indeed, who could say whether the sterilization of the biliary passages was really due to the drug? Might it not have followed efficient surgical measures, including drainage? Still, the report is worth noting, and sulphanilamide might well be tried on typhoid carriers when they present themselves.

[1] *The Journal of the American Medical Association*, July 9, 1938.

[2] *The Canadian Medical Association Journal*, June, 1938.

Abstracts from Current Medical Literature.

MEDICINE.

Some Unsolved Problems of Tuberculosis.

James Alexander Miller (*The Journal of the American Medical Association*, July 9, 1938) draws attention to the fact that though research has greatly increased our knowledge of tuberculosis, there are many unsolved problems related to the pathogenesis and epidemiology of this disease. The relation of resistance to infection has not been determined, but there is evidence that resistance, both natural and acquired, has an important role in modifying the liability to disease after exposure to infection. One of the most actively debated questions is how does phthisis develop? One school of thought believes that it is produced by exogenous reinfection or by first infection in adolescence or adult age. The majority of careful students, however, accept the view that the development of phthisis is usually due to exacerbation of a preexisting lesion, the exacerbation being especially dependent on variation in resistance; that there is a long latent period before the lesions become exacerbated; and that in the great majority of cases they never develop into clinical disease. The study of resistance is probably of greater importance than the study of infection. There is a sharp difference of opinion as to whether it is safer for the individual to react or not to react to tuberculin. Periodic repetitions of tuberculin surveys may, by drawing attention to the changing reactions, give valuable information regarding incidence of exposure to infection and relationships to clinical disease. Radiological surveys are demonstrating thousands of cases of latent tuberculous lesions. The proper management in these cases is one of the most important present-day problems. Finally, the author demands careful consideration of what is the ultimate goal aimed at in the control of tuberculosis. Total eradication of the disease may result later in acute epidemics of tuberculosis. Some form of vaccination, either artificial or by doses which are overcome by the body resistance and which are similar to the subclinical infections incurred throughout the civilized world, may be the best. The author adds that it is certainly a cardinal mistake to assume that the methods now employed are sufficient to solve the tuberculosis problem.

Thrombophlebitis of the Leg.

G. de Takáts (*The Journal of the American Medical Association*, April 2, 1938) states that the principal aim in the treatment of deep-seated thrombophlebitis of the leg is the prevention of embolism, of ascending thrombosis and of persistent œdema. From the day of the formation of the clot every effort must be made to rid the limb of the swelling, as the persistence of fluid rich in protein will produce permanent fibrosis in the subcutaneous tissue. To this end fluids are restricted to one litre per day; salt is restricted, and ammonium chloride is given in doses of 1·0 gramme (15 grains) four times a day. The limb is raised as high as possible and a heat cradle is applied. Two or three days later a mercurial diuretic is given intravenously. The intravenous injection of calcium gluconate has also proved helpful. All these drugs help to mobilize fluid from the œdematous limb; it is much easier to prevent further swelling by elastic support after the œdema has subsided than to treat it if the patient gets out of bed with a swollen limb. When the temperature has become normal and the pulse rate has remained normal for ten days or so, it is usually safe to allow sitting up, slight active exercises and a few steps. Should the pulse rate rise again, further immobilization is necessary. The sedimentation rate, if estimated, is an excellent guide to the activity of the clot. After the patient gets out of bed elastic support must be maintained for a year or longer.

The Rash in Meningococcal Infection.

C. V. Craster and H. Simon (*The Journal of the American Medical Association*, April 2, 1938) point out that the rash of meningococcal infection is petechial, and when the condition is fulminating a purpuric rash accompanies the petechial rash. The rash occurs before the cerebro-spinal fluid becomes cloudy and before meningism appears; that is to say, during the initial meningococcæmia. They recall the observation of McLean and Coffey, in 1931, that in four cases out of five, smears showing intracellular meningococci might be obtained by lightly puncturing a petechia or a purpuric patch and staining the resultant drop of blood, smeared on a glass slide, by Gram's method.

Electric Arc Welders.

N. Enger and O. A. Sander (*The Journal of Industrial Hygiene and Toxicology*, May, 1938) describe chronic lung changes in men engaged in electric arc welding. X ray examination of the lungs of 26 men who had used electric welders for an average period of nineteen years revealed the presence in five of nodular shadows which resembled those of silicosis. Examination of five other men showed exaggerated trunk shadows. The past history of these patients revealed no exposure to silica, and it was concluded that welding with bare metal electrodes had produced these lung changes. Those in whom these X ray signs were present had worked inside steel tanks, welding the seams, for long periods. These tanks had only two openings, one measuring 27·5 by 45·0 centimetres (eleven by eighteen inches), the other 7·5 centimetres (three inches) in diameter; hence the ventilation inside them was very poor. None of these men complained of any symptoms, nor did clinical investigation reveal any evidence of disease. A *post mortem* examination on one man who had died of pneumonia was carried out sixteen months after death. There was some evidence of emphysema, and pronounced staining with Prussian blue indicated that the nodular opacities were due to the deposit of iron oxide. The iron oxide was derived from fumes. The iron particles were in such fine suspension in the inhaled fumes that the majority were seen to be in Brownian movement when attempts at counting them were made, being less than 0·5μ in diameter. Iron was deposited also in the lymphatics, but there was no evidence of fibrosis in the lungs. There was no evidence of functional impairment of the lungs.

The Diagnosis of Subclinical Scurvy.

Ruell A. Sloan (*The Journal of Laboratory and Clinical Medicine*, July, 1938) compares the methods used in the detection and grading of cases of subclinical scurvy. Sixteen scorbutic patients, two non-scorbutic patients with bleeding, and five normal persons were examined by all available methods. Bedside diagnosis is difficult, as the signs and symptoms are vague and are frequently masked by complicating disease. The occurrence of hæmorrhage was found to be the most helpful sign, bleeding gums and epistaxis occurring most commonly; but gastrointestinal hæmorrhage, menorrhagia, subdural hæmatoma and petechial hæmorrhages have each occurred as the only clinical sign in the author's cases. The capillary resistance test is the simplest diagnostic procedure and may be performed at the bedside. In the majority of cases it gives dependable information concerning vitamin *C* depletion. A drawback to this test lies in the occurrence of falsely negative results due to severe anæmia. The chemical tests appeared somewhat more sensitive to very mild deficiencies than the capillary resistance test. The estimation of the vitamin *C* content of the blood was considered to be the simplest dependable procedure and roughly indicated the degree of depletion. It was found that the estimation of a single sample of urine was a rough and frequently false measure of vitamin *C* saturation. The author considers that the most precise and dependable method of determining the degree of vitamin *C* saturation is the determination of the rate of absorption of an injected dose from the blood stream. The rate of excretion of a test dose is less precise, but quite dependable. On account of their greater dependability it is probable

that these methods will supplant the capillary resistance test, though with some experience the latter makes the recognition of the disease simple in the majority of cases. If the readings are made during a course of treatment, and an appreciable recovery in capillary resistance occurs, definite information is gained regarding the deficiency in vitamin *C*.

The Ætiology of Chronic Dyspepsia.

A. B. Rivers and A. E. M. Ferreira (*The Journal of the American Medical Association*, June 25, 1938) have analysed the case histories of some four thousand patients who were investigated for dyspepsia at the Mayo Clinic, and about half of whom had presented themselves for treatment of this complaint. There was a slight preponderance of males in the whole series, but this varied in the different age groups. The age range was from fifteen to sixty years. For purposes of this classification the following types of dyspepsia were distinguished: organic, referring to diseases of the tissues of the stomach and duodenum; reflex, from disease in the gall-bladder, biliary ducts, appendix, pancreas or intestine; systemic, due to faulty metabolism, toxæmia, deficiency diseases or dysfunction of the organs of internal secretion; functional, to include disturbances in normal gastro-duodenal activity not resulting from demonstrable disease elsewhere in the body. Of the whole series, 37% fell in the systemic group, 25% were functional, 22% were due to reflex causes, and only 14% were attributed to organic disease of the stomach. The causes of dyspepsia as a primary complaint, in order of importance, were found to be as follows: functional causes, peptic ulcer, cholecystic disease and cancer of the stomach. These were responsible for nearly two-thirds of the cases. The relative incidence and causes of the symptom for males and females are presented in three age groups: fifteen to twenty-four years, twenty-five to thirty-nine years, and over forty years. Chronic appendicitis was responsible in only 2·2% of the series, and the majority of the patients were under the age of twenty-five years. It is therefore suggested that appendicectomy as a treatment for dyspepsia in patients over the age of twenty-five years is not likely to produce a satisfactory cure. The authors were not surprised to find that the highest incidence of functional dyspepsia was between the ages of twenty-five and thirty-nine years. After the age of forty years the relative importance of the functional basis became less, as by this time diseases of more significance had developed. Peptic ulcer was the lesion most frequently found in men over thirty-nine years of age who came for examination for indigestion. It was four times as common among men as women. Among women, cholecystic disease was second only to functional disturbances as a cause of dyspepsia. It was found to be the cause in every fourth woman over forty years of age suffering from dyspepsia. Although not the most common among the causes of indigestion, cancer of the stomach is easily the most important. Failure to recognize it is a fatal error. The incidence in this series was 3%. One man in every six over the age of forty years, complaining of indigestion, had cancer of the stomach. The importance of educating the layman to realize the risks he may incur by treating his own indigestion according to the advertisements of any vendor of therapeutic nostrums is emphasized.

The Nature of the Bleeding in Jaundice.

Armand J. Quick (*The Journal of the American Medical Association*, May 14, 1938) has devised a simple procedure for the determination of prothrombin in blood. It depends on the observation that the clotting time of oxalated plasma, to which has been added an excess of thromboplastin and an optimum amount of calcium, can be employed as a direct measure of the prothrombin content of the plasma. The importance of this test lies in the fact that, of the four main agents required for the clotting of blood, namely, thromboplastin, prothrombin, calcium and fibrinogen, only the latter two have been closely observed in cases of hæmorrhage. Now that a simple method for the quantitative determination of prothrombin is available, evidence has been obtained to show that a close relationship exists between prothrombin deficiency and the severity of the bleeding tendency in jaundice. There are three ways of producing experimental prothrombin deficiency: by withholding vitamin *K*, by the giving of a toxin (present in spoiled sweet clover hay), and by injury to the liver. When the prothrombin level falls below 20% of normal a definite hæmorrhagic tendency is always observed, and if the level can be raised normal clotting will take place. Investigation of a number of jaundiced patients has demonstrated the clinical fact that only a few of these patients suffer from a hæmorrhagic tendency; it is in these few cases that a low prothrombin level in the plasma exists. It is interesting to note that there is a wide margin of safety, nearly 80% of the normal amount of prothrombin being lost before the clotting time is increased. It is of importance to determine clinically in any case of jaundice in which operative interference is being undertaken, whether any liver damage has occurred. The synthesis of hippuric acid is suggested as a useful test for this purpose. If the result of this test is unsatisfactory, operation should be undertaken only as a matter of urgency. Dextrose and gelatine (substances with a high amino-acetic acid content) are said to be useful in building up the liver reserves. In the presence of prothrombin deficiency, vitamin *K* (as in powdered alfalfa leaf) should be administered with bile and bile acids to assist the absorption. If the prothrombin level is already below 15%, immediate blood transfusion is the only treatment. This will be of only temporary benefit, the guide to its repetition being the prothrombin level. It is possible to maintain this level above the bleeding point by blood transfusion while other methods of treatment are being employed to restore the body's power to produce prothrombin.

Treatment of Pellagra with Nicotinic Acid.

Jean M. Grant, Elizabeth Zschiesche and Tom D. Spies (*The Lancet*, April 23, 1938) have reported the successful treatment of pellagrins maintained on a pellagra-producing diet. Seven patients were studied; all had extensive lesions of the mucous membrane, six showed characteristic skin changes, four had mental symptoms, and four had peripheral neuritis. They were restricted to a pellagra-producing diet, and when no healing of the mucous membrane lesions was observed after two or three days, treatment was instituted. Supplements of 200 to 1,500 milligrammes of nicotinic acid per day were given, divided into doses of 100 to 200 milligrammes. The condition of the mucous membrane of all patients improved within forty-eight hours, and usually improvement was noted within twenty-four hours. The ptyalism, Vincent's angina and increased porphyrinuria likewise disappeared, but the peripheral neuritis was not cured. Appetite improved, the sense of apprehension was lost and the patients acquired a sense of well-being. Symptoms recurred in two patients maintained on the pellagra diet when the nicotinic acid supplements were discontinued.

Richard France, Robley D. Bates, junior, W. Halsey Barker and Edward Matthews (*Bulletin of the Johns Hopkins Hospital*, July, 1938) report two cases of moderately severe pellagra in which nicotinic acid was used, in the one orally and in the other by intramuscular injection. Prompt healing of all the lesions took place in both cases. These authors stress the low cost of this treatment of pellagra in contrast with the earlier forms of therapy. Post-operative glossitis (in one patient with pyonephrosis and in one who had been subjected to gastroenterostomy) healed rapidly under oral therapy with nicotinic acid. The authors recommend the intramuscular use of nicotinic acid, both therapeutically and prophylactically, in the treatment of patients whose intake of food is by necessity very limited, as in cases of persistent vomiting, anorexia or post-operative states following major surgical procedures involving the gastrointestinal tract.

Proceedings of the Royal Commission Appointed to Inquire into Matters Pertaining to National Health Insurance.

Monday, September 19, 1938.

Mr. W. R. Gilbert was recalled and further cross-examined by Mr. Abrahams concerning the operation of the institute of which he is secretary.

Mr. Abrahams: I understood you to say on Friday that this institute is paid so much per member by the lodge?

A.: That is so.

Q.: The member pays into the lodge and the lodge pays your institute in respect of the member for medical services?

A.: That is correct.

Q.: The rate is 22s.?

A.: For a full-rate member.

Q.: What are the advantages to the member in belonging to your institute as distinct from remaining with the lodge for medical attention?

A.: They obtain the service of our hospital, and in addition there is the service of our nurse at our rooms, all at a slight cost to the lodge, which we hope is passed on to the member.

Q.: I suppose that is a great advantage?

A.: Certainly, considering the low schedule operation fees also.

Q.: All of these things ought to be very attractive?

A.: Yes.

Q.: Unless there was some counterbalancing disadvantage?

A.: That is so.

Q.: One would think that members would flock to you?

A.: Yes.

Mr. James Reid was examined by Mr. Dovey: You are an accountant practising in Brisbane and you are the Secretary of the Queensland Friendly Societies' Association and the Friendly Societies' Medical Hospital Council?

A.: Yes.

Q.: I understand that the association consists of the different friendly societies who have lodges in Brisbane and in the suburbs of Brisbane?

A.: Yes.

Q.: And Queensland generally?

A.: Yes.

Q.: Can you tell the Commission how contracts are made with medical practitioners in Brisbane for medical service to friendly society members?

A.: In the first place the Friendly Societies' Medical Council contracts with the British Medical Association doctors. A doctor who makes application to receive members on his list must first make his application through the British Medical Association, who approves of his application and forwards it to the council for its acceptance. A form of agreement is provided, which is signed by the doctor.

Q.: During the taking of evidence before this Commission some medical practitioners have produced forms of contract in which the parties are the medical practitioner on the one hand and the individual lodge on the other. Are there many British Medical Association doctors working in Brisbane and the suburbs who work under any agreement other than that?

A.: Not to my knowledge; that is the practice in respect of contracts.

Q.: There are a number of practitioners in Brisbane, of course, who are not members of the British Medical Association and who do contract practice?

A.: Yes.

Q.: Do you know how they effect contracts, and with whom?

A.: In that case they deal directly with the friendly society lodges concerned.

Q.: Does the council have any control over the friendly society institute?

A.: None whatever.

Q.: Do you work in harmony with those who control the institution?

A.: So far as the friendly societies are concerned, definitely yes; but so far as the medical practitioners are concerned, they work on their basis and we have no conflict with them.

Q.: I suppose you are able to tell the Commission that some members of the friendly societies throughout Brisbane go to the institute for their medical benefit and others go to private practitioners?

A.: That is so.

Q.: Take the case of a lodge member who gets his medical service at the institute; would not it cost the lodge less; who gets the benefit of that?

A.: In that case I should say the individual member himself.

Q.: As far as your council is concerned, how is the capitation fee payable to the doctors?

A.: It is a fixed payment, under an agreement made between the British Medical Association and the Friendly Societies' Medical Council, which was arrived at on August 24, 1934.

Mr. Reid then explained the manner in which the capitation fee was arrived at. In 1934 the capitation fee was 25s., and by arrangement with the British Medical Association it varied as the Commonwealth Statistician's nominal wage index number varied, the fee now being 26s. *per annum*. Mr. Reid then detailed the fees paid by the various classes of members and gave the numbers of members in each class, but was unable to give any estimate of the number of dependants for each member paying full rates. He stated that he understood information on the point was being prepared by the Protestant Alliance Friendly Society.

Mr. Dovey: Is the scope of the medical service to be provided set out in the common form of agreement?

A.: We regard the agreement as covering the scope of service. We have issued to all members who are on the list through the council and to the doctors connected with the council, a book of rules which sets out the points connected with the agreement, and also have a leaflet termed "Notes to Lodge Doctors re Medical Service for which payment is to be made". We set out whether the lodge member pays ordinary or reduced fees for all those illnesses or injuries excluded by the agreement, and he has a clear-cut issue as to what he has to pay for.

Q.: Are there many disputes or complaints?

A.: Very few indeed.

Q.: Does rule 17 mean that if excluded services are rendered by the lodge doctor to the patient and not paid for, you apply sanctions to the extent that he cannot get included services?

A.: That is provided, but it is not carried out to any extent. It is really to protect the medical officer.

Q.: I understood you to say that the issuing of these instructions had reduced the number of causes for difference between the patient and the practitioner?

A.: That is so.

Q.: Can you say whether or not, from the point of view of lodges, you get satisfactory service from the medical practitioners?

A.: Oh, yes; it is generally conceded today as being a very satisfactory service.

Q.: You desire to say something about reference number 7 of the terms of reference to this Royal Commission?

A.: There is just this aspect: should the payment for services rendered outside the contract be a matter of arrangement between the patient and the doctor or be defined in the contract to indicate, first, the rate of such payment and, secondly, the manner in which it should be secured? The opinion of this council is that if the method is defined in the contract to indicate, first, the rate of

such payment and, secondly, the manner, the friendly societies here consider that that would be advantageous. We already include in our rule books a list of operation charges.

Q.: You have a provision which sets out the scale of fees which under the contract medical practitioners are to be allowed to charge and receive for excluded services?

A.: That is so.

Dr. Mulvey: Who do you suggest should collect the fee, the doctor or the Commission?

A.: At present it is the doctor, and I think that system should be continued.

The Chairman: I do not think it is contemplated that the Commission should collect these fees for outside services.

Mr. Dovey: I think one witness suggested that insurance practitioners should be allowed reasonable fees for excluded services and should be assisted as far as possible to collect them. How it is to be done is difficult to see, in view of the fact that there is nothing in the Act, nor can there be anything in the regulations to cover the point. Perhaps the approved societies could do something to help. In respect of fees for excluded services, does your council do anything to assist the doctor to collect such fees?

A.: In glaring instances permission is withheld to transfer from one doctor to another when the member owes money to the first man.

Q.: In the case of night fees which remain unpaid, is any attempt made to enforce their payment either by the council, the association or the individual lodges?

A.: If a dispute arises the council compels the member to pay in accordance with his agreement.

Mr. Reid stated that the Friendly Societies' Council considered that the existing arrangement between the British Medical Association and the council should be continued for those members who did not come under the national scheme.

The Chairman: There are two classes of case. I understood the witness to speak of the member who does not come under national insurance, and he suggested that in his case the present system should continue. Suppose he comes under the national insurance scheme and is liable to pay 1s. 6d. per week, but has dependants also. What do you think should be done about those dependants? Do you suggest that they should get the benefit of the wider scope of service, or should it be limited to the scope now rendered?

A.: A wider scope based on their capacity to pay. The friendly society member has been educated to what we call a good service. The married member is concerned largely with what provision is to be made for his wife and children.

Q.: Then if he is an insured person paying 1s. 6d. a week you think he would probably prefer to pay some slight additional amount, say 4d. or 6d. a week, so as to get the wider service for his dependants?

A.: Yes. He will get the benefit of the fact that the friendly societies will be able to reduce their fees to some extent after the introduction of national insurance.

Mr. Abrahams: What does the adult married man pay now for all of the benefits he receives?

A.: The rate varies according to the society and to the age group of the member when he joined. It might average from 1s. 6d. to 2s. a week.

Q.: The married man is paying that rate now for himself, his wife and children, if he has any?

A.: Yes.

Q.: Do you think that the average friendly society member will be able to afford to pay an increased fee in addition to the 1s. 6d. a week for national insurance?

A.: That is largely a matter for the individual. The average friendly society member would, I think, be prepared to pay a little more for a better service.

Mr. Charles Edward Chuter was examined by Mr. Dovey: I understand you are Permanent Under Secretary, Department of Health and Home Affairs?

A.: That is so.

Q.: The Department of Health controls, in the State of Queensland, a number of hospitals among other things?

A.: We administer the hospitals law for the State.

Q.: Quite recently the system of finance and of administration of the hospitals generally in the State, and in some parts of the State particularly, has been changed?

A.: Yes. It has been a gradual change. It was brought about to deal principally with the position of the hospitals in Brisbane.

Mr. Chuter then gave detailed evidence as to the administration of the hospitals in Queensland and the districts into which the State has been divided. After further statistical evidence Mr. Chuter was examined concerning the change from the honorary system.

Mr. Dovey: We have been told that in respect of northern Queensland ordinarily in the case of industrial awards a special allowance is made for climatic and other conditions. In the case of appointments to hospitals is there any allowance made for the fact that they are situated in the tropics?

A.: No. Of course, the salary is at a fixed rate, and that does not come into it at all. It has never been considered.

The Chairman: Does not that influence the amount of the salary, that is, the fact that a man has to go right away? Would a man in Townsville, for instance, receive only the same rate as a man in a district further south?

A.: I think the rate of salary is based on the size of the hospital; I do not think they have the slightest difficulty in filling the senior positions.

Mr. Chuter was further questioned, mainly as to various statistics which he said he would furnish to the Commission when available. He was then questioned by Dr. Mulvey and the chairman as to the hospital system in Queensland generally.

Dr. Mulvey: I understand that you have some difficulty in getting applicants for salaried positions as superintendents of hospitals in small towns?

A.: In the far outpost places chiefly.

Q.: And for assistants in the larger towns?

A.: Yes, junior appointments; there has been some difficulty.

Q.: Do you give any encouragement to general practitioners to carry on their work at the hospitals in those places?

A.: Only where what we know as the honorary medical system is in operation. There may be one or two places in which the whole of the medical practitioners are allowed to enter the hospital.

Q.: It is not your policy to discourage the development of the general practitioner service in those country districts?

A.: We have not done so.

Q.: By having superintendents and general staffs of the hospitals in salaried positions, is there not the danger that the general practitioner is discouraged from carrying on his ordinary work?

A.: Actually the position in this State is that the hospital authority is allowed to decide what medical organization it desires. For instance, there have been places where the honorary medical system has been abolished and paid staffs have been substituted.

Q.: What effect does that have on the general practitioner in the district?

A.: I think it has some effect on him because the use of the hospitals where that has happened has increased very considerably.

The Chairman: The number of patients has increased?

A.: Yes, both in-patients and out-patients.

Dr. Mulvey: Consequently the general practitioner loses a considerable portion of his ordinary work?

A.: That would be the case. Innisfail is a case in point. I can give you figures relating to when the honorary system was in operation and since it has been abolished. Another case is that of Gympie. I shall see if I can obtain the figures in regard to it.

The Chairman: What has been the effect on the general practitioners in the district where that system has prevailed?

A.: I think it has had some effect upon them, because there have been protests against the system from the

doctors; but the people have not seen fit to alter the system notwithstanding the protests.

Q.: Are they free to exercise their own discretion apart from any government control or suggestion?

A.: Yes, they are generally allowed to exercise their own discretion.

Dr. Mulvey: Do you give them a bias or indicate the policy you have in view?

A.: I do not know about that; sometimes we express an opinion if we are asked to do so.

Q.: In ten or fifteen years' time what will be the role of the general practitioner?

A.: I am inclined to think that the paid system will gradually become greater and greater. That seems to be the general trend in this State. As a matter of fact, approximately 50% of the births in this State now take place in public institutions.

The Chairman: Attended by the salaried staff in most cases?

A.: In the public hospital by a salaried staff and in an intermediate by their own practitioner, except in the one or two cases I have mentioned.

Mr. Abrahams: Is there a Government representative on each of these public hospitals?

A.: Yes. In the case of the hospital boards the Government generally has five representatives and the local authority or local authorities four. In some places there is a contributors' representative. In a case like that the Government would have four, the local authorities four, and the contributors one. Those contributors are really persons who are contributing to a benefit scheme.

Mr. Dovey: The Government representatives are local people of standing nominated by the Government?

A.: That is so.

The witness then withdrew and the Commission adjourned at 4.30 p.m. until Tuesday, September 20, 1938, at 10.30 a.m.

Tuesday, September 20, 1938.

Mr. Robert Leslie Avery Hill, an executive officer of the Associated Friendly Societies of Queensland, was examined by Mr. Dovey.

Mr. Dovey: Are you able to speak of the feeling of members of your organization regarding the provision of medical services for dependants under national health insurance?

A.: Yes.

Q.: Tell us what the position is so far as your organization is concerned?

A.: I have interviewed my lodge members from Brisbane as far up as Bowen, and the general consensus of opinion among them is that in respect of national health insurance they are mostly concerned about medical and chemist's service for their dependants. That is the general opinion not only among members of friendly societies. For instance, I addressed a meeting in Gympie last Saturday week, which was attended by over 100 people, and the general consensus of opinion among them also—and it was not a friendly society meeting—was that they required a service for their dependants.

The Chairman: The same service as the insured man would get under national health insurance?

A.: At the present time the public do not quite understand the services which will be applicable under the scheme. The majority of the people in Queensland today are used to friendly society service; so far as members are concerned and so far as doctors are concerned we are a happy family and the service is really good. We are working under a contract today which falls in with the idea of every member, and the main object of friendly society members is to retain that same service for their dependants.

Mr. Dovey: And what is their view of the services to be given under national health insurance?

A.: They are perfectly satisfied with it; but if it is within the power of the Government to extend such a service, I am of opinion that present-day friendly society members would be prepared to pay for a slight extra service under the scheme. At present, under lodge practice, a man might go to a doctor and say: "I do not know my complaint." When we go to a general practitioner we should be in a position to seek pathological examination or even X ray examination. The average lodge member, to use an Australian term, bucks when he has to be forced on someone else for pathological service. . . .

Mr. Abrahams: I want you to elucidate one or two points which you have just mentioned. When national insurance comes in there will be certain friendly society members who will not come under the scheme; the Commonwealth Government, as you know, estimates that number at one-third, and the other two-thirds are expected to come under the scheme. The workers will come under the scheme, but the families, as you know, will not. In theory that will set free the wives and children for private practice, that is, in theory. I gather that your view is that those who can afford it will want their wives and children still to be covered by friendly societies?

A.: That is so.

Q.: They will not be private patients, but contract patients?

A.: Yes.

Q.: And from your experience you believe that will be the position of those who can afford it?

A.: Yes.

Q.: I suppose there will be some of your members to whom the 1s. 6d. a week, which they will have to pay under the scheme and which covers all sorts of things besides medical benefit, such as pensions and so on, will be a major financial consideration?

A.: I think it would only be a few.

Q.: You think, for instance, that everybody will be able to afford 1s. 6d. a week and then something for a lodge? I want your candid opinion?

A.: The matter is largely psychological. Various governments in the past have imposed taxes, such as the relief tax. When the tax was imposed in Queensland it did not have any detrimental effect on the friendly society movement. Again, today, so far as that 1s. 6d. for national insurance is concerned, you will have that same psychological effect—"for what the eye won't see the heart won't grieve". From conversations which I have had with members of various friendly societies I gather that they will continue with their friendly society, because friendly societies today are offering the best workers' insurance in the Commonwealth.

Mr. Vivian Birt, assistant secretary of the Ancient Order of Foresters Friendly Society, was examined by Mr. Dovey concerning the likelihood that lodge members would continue to arrange for medical services under the friendly society lodge system.

Mr. Dovey: What do you say so far as they [the members of Mr. Birt's society] are concerned?

A.: My society is concerned as to the effect of national health insurance on our present members, and we desire to be in a position to continue to provide medical services for wives and dependants. We have been informed that the national health insurance medical service for the insurees is to be wider in its scope than the present friendly society service. This is very desirable if it is within the willingness to pay. We would welcome this wider scope to our members' wives and families only if it entails no increase in present contributions, or if any such increase is offset by a government subsidy.

Q.: I think that is where, in your opinion, the feeling among your members differs from that which Mr. Reid told us. Mr. Reid told us that in his opinion members would be prepared to pay a slight increase in order to get a wider service?

A.: I do not believe that that is so.

Q.: Is that based on your contact with individual members?

A.: Yes, it is based on my personal contact with at least 2,000 of my members. Most of our members are now asking what reduction in their present contribution will result when national health insurance comes into force,

and although they are asking for a continuation of the present service for their wives and families, they are not pressing for any wider scope of such service, beyond the present friendly society service, if it means increased contributions.

Mr. Birt stated that it was hoped by his society that their members' contributions would be reduced upon the introduction of national health insurance, and he was then cross-examined by Mr. Abrahams on this aspect.

Mr. Abrahams: You mean that part of the 1s. 6d. which fairly represents a man's payment to his lodge for his own medical and chemists' services?

A.: Yes. We hope that that will be definitely reduced on January 1, not on April 1. The date on which medical benefits will be commenced will be April 1, not January 1.

Q.: What you say is: "Here is a married man who is now paying 1s. 6d. a week for himself, his wife and his children; he will now have to pay 1s. 6d. for himself alone, and we are in hopes that he will only reduce his payment to the lodge by that part of the 1s. 6d. that he now pays which represents the service that he himself now gets from the lodge"?

A.: That is so.

Q.: Supposing we assume that amount to be 6d.; you hope that he wil continue to pay the balance of 1s. for his family?

A.: That is why I am here today.

Q.: But a single man and perhaps the unmarried female will drop out altogether?

A.: No, they will not drop out of the lodge altogether; they will go what we call non-medical.

Q.: So you will lose his contribution as far as medical service is concerned?

A.: That is so.

Q.: You have said that so far as you can ascertain from conversation with 2,000 members, you want the extra service if it be within their willingness to pay?

A.: Yes.

Mr. Birt was questioned concerning the contributions a man under the national health insurance scheme would have to make to a friendly society in order to provide for medical attention to his dependants.

The Chairman: If you cannot assess the amount, make a guess. You say that 1s. would not be enough. What would be enough?

A.: I would guess the amount at approximately 1s. 4d. a week; that would mean a reduction of contributions by 2d. a week.

Q.: Explain what that 4d. a week is for?

A.: For the doctor and chemist, for the wife and family.

Q.: Then you think that the man who is paying 1s. 6d. per week for insurance for himself, if he desires to get medical attention and chemist's attention for his wife and family, would be required to pay another 1s. 4d. a week?

A.: That is about it.

Q.: Assume that for the moment; do you say that your members would not be willing to continue to pay that 4d. a week over and above the 1s. 6d. a week they would have to pay under the national health insurance scheme?

A.: They would pay that onto their non-medical rates of contributions. What I have been trying to stress is that members will not continue in the lodge unless there is some reduction of their present total contributions.

Q.: That may be; instead of paying their present contribution of 1s. 6d., it would have to be reduced to 1s.; instead of paying 1s. 6d. insurance *plus* 1s. 6d. lodge, they would have to pay 1s. 6d. insurance *plus* 1s. lodge, in order to protect their wives and dependants?

A.: If it could be arranged for 1s. I am sure that 95% of our members at least would continue.

Mr. Abrahams: The difficulty is that benefits other than medical benefits cost more than 10d. You have put it that the full benefit, including payment for the dependants, would be 1s. 4d.?

A.: That was just a guess; I could not support it.

Q.: You work it out that, assuming a payment of 4d. for the doctor and chemist for the wife and children, the lodge member would still have to pay 1s. 4d. instead of the present 1s. 6d.?

A.: Yes, which I feel they would do.

Q.: You feel that if they were given an allowance of 2d. a week they would pay it?

A.: That is so.

The Chairman: Then that means that the man who has to pay both insurance and lodge will be prepared to continue both if, instead of having to pay 3s., he had to pay 2s. 10d.?

A.: Yes. My reason for saying that is that medical benefit is not the entire friendly society service. There are other aspects of friendly societies; there is the sick pay, which is still payable from the first day of sickness; there is the funeral benefit for the member and his wife; and there is the entertainment and social side of the meetings.

The witness then withdrew, and at 12.35 p.m. the Commission adjourned to Number 1 High Court, Darlinghurst, Sydney, on Thursday, September 22, 1938, at 2.30 p.m.

Thursday, September 22, 1938.

Proceedings were conducted *in camera*.

Friday, September 23, 1938.

Dr. Ralph Clifford Huntley, Mosman, was examined by Mr. Gain. He stated that he had had experience during 1935 as a *locum tenens* in a panel practice at Hull, in England. The fees were 2s. 6d. to 3s. 6d. for consultations and 3s. to 5s. for visits, both including medicine.

Mr. Gain: What was the type of equipment at the surgery?

A.: It was very poor.

Q.: What about instruments?

A.: You would not call them instruments, practically.

Q.: Did you notice anything as to the treatments of any ailments or difficult illnesses in that specific panel practice?

A.: Well, the chief idea seemed to be just to do the ordinary bread and butter work; they were treated, but if there were anything liable to lead to a complication or in any way give the doctor any work or worry they were sent to the infirmary.

Q.: Did you notice anything about the attitude of the patients themselves as to the treatment they should get?

A.: They did not get a say in what they expected; they just took it. If they objected you did not listen to them; they were just fired out, asked to move on. They did not even take their hats off; there was no time. They had no come-back. You would say "Next please" and they would go out of the door.

Q.: Were you able to form any opinion as to the standard of treatment given to panel patients compared with the treatment given under the contract practice here?

A.: I do not think there is any comparison if a man is conscientious in his work. Over there the doctor rules the people. Out here the people rule the doctor. You make a conscientious effort to please the people.

Dr. Huntley gave further evidence concerning his experiences of panel practice and was cross-examined thereon by Mr. Dovey and Mr. Williams.

Dr. Eric Hilton Miles, Sutherland, was examined.

Mr. Abrahams: I point out to the Commission that this doctor has no figures, but he lives in a peculiar locality, which necessitates his taking a rowing boat in order to get to his patients across a stretch of water. His case illustrates what was said this morning, that it is probably desirable to have the mileage as an excluded service, as no special case of this sort can be dealt with in the contract. All of this doctor's patients are within the three-mile limit, and his case points to the desirability of some such arrangement as mileage being outside where a special contract could be made as in this case, or in some areas where special circumstances, such as the extraordinarily bad state of the roads, as in north Queensland, where the Commission would have the power by the specially excluded service to make a special arrangement applicable to the special circumstances. That is what we are going to suggest.

Mr. Gain: Your locality is very mixed, partly residential, partly industrial, and to some extent a holiday area, and it embraces Port Hacking, Cronulla, National Park and numerous bays around Port Hacking?

A.: Yes.

Q.: And the areas you serve are Jannali, about one mile away; Como, two miles away; Miranda, two miles away; Gymea Bay, two and a half miles; Oyster Bay, two to three miles; Engadine, four to five miles; Sylvania and Caringbar, four miles; Taren Point, four to five miles; Heathcote, six miles; Menai, six miles; and Waterfall, nine miles?

A.: Yes.

Q.: Describe to the Commission some of the places you have to visit around the foreshores, and how you get there?

A.: In certain areas you drive your car to perhaps a half or three-quarters of a mile from your destination and then scramble over rocks and climb down steps. Some of it knocks you out quite a lot; there may be 80 or 100 steps.

Mr. Dovey: Are those places not served by tradespeople?

A.: Yes.

Mr. Gain: What about climbing up the steps?

A.: I have to sit down for about ten minutes to get settled down again.

Dr. Miles gave further details concerning difficulties of access to patients in his district, and generally as to the difficulties of transportation, and after cross-examination by Mr. Dovey, withdrew.

The Commission adjourned at 4.48 p.m. to Monday, September 26, 1938, at 10.30 a.m.

British Medical Association News.

NOMINATIONS AND ELECTIONS.

The undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Pearson, Herbert Hilary, M.B., B.S., 1938 (Univ. Sydney), Western Suburbs Hospital, Croydon.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Armati, Roy Edgar, M.B., B.S., 1937 (Univ. Sydney), The Gladstone, Gladstone Road, Leura.

Dryer, Albert Thomas Benedict, M.B., B.S., 1938 (Univ. Sydney), Parramatta District Hospital, Parramatta.

The undermentioned has been reelected a member of the New South Wales Branch of the British Medical Association:

Hynes, Marie Eleanor, M.B., B.S., 1928 (Univ. Sydney), 704, Pacific Highway, Killara.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A meeting of the Melbourne Pædiatric Society was held on June 8, 1938, at the Children's Hospital, Carlton, Dr. Colin Macdonald, the President, in the chair.

Bronchiectasis.

Dr. L. P. Wait, on behalf of Dr. W. W. McLaren, showed a girl, aged nine years, suffering from bronchiectatic dilatation of the base of the left lung, which was localized to the extreme left posteriorly. She had had an attack of bronchopneumonia when only four months of age, and at eight years of age had left basal pneumonia. She had had a persistent cough from early infancy; but for the past year the cough had been more troublesome, and each morning she coughed up several drachms of mucopus. She had been in hospital for three weeks; and though she was well nourished, rhonchi were audible at the left base. With posture treatment the amount of sputum had increased in the early stages and decreased later. Dr. Wait demonstrated the extent of the lesion in skiagrams made after instillation of lipiodol, and asked for opinions concerning (*a*) the prognosis without operation and (*b*) non-surgical treatment. If an operation was considered desirable, Dr. Wait asked for advice as to the time when the operation should be performed, the preoperative treatment and the choice of anæsthetic. The mortality rate might be as high as 64%.

Dr. Howard Williams, on behalf of Dr. Ian Wood, also showed a patient with bronchiectasis. The girl, aged twelve years, had had a cough from the age of six months, from which she had never been free. She had had diphtheria and mild measles and slight pertussis in early childhood, but had not had any evidence of pneumonia. The child was rather pale, and exhibited early clubbing of the fingers and toes. Some harsh bronchial breath sounds were heard at the left base, with impairment of the percussion note. The amount of sputum was less than 11 cubic centimetres (four drachms) per day of non-offensive, though mucopurulent, material, from which diphtheroids and pneumococci had been cultured. The hæmoglobin content of the blood was estimated at 105% (Sahli) and the erythrocytes numbered a little over five million per cubic millimetre. Dr. Williams showed in skiagrams made after the introduction of lipiodol that the lower lobe of the left lung was collapsed, and that early bronchiectatic changes were present in the right lower and middle lobes in addition to advanced bronchiectasis in the left lower lobe. The object of presenting the patient was to get an expression of opinion on the prognosis, and comments on the advisability of surgical treatment and the type of operation which should be considered.

Dr. Williams opened the discussion with a brief account of the history of bronchiectasis. The condition had been known pathologically for many years. It was convenient to divide the historical discussion into a consideration of three eras: the first the pathological era, the second the era of accurate clinical diagnosis, and the third the era of modern surgical treatment.

He said that the pathological era embraced the later part of the eighteenth century and the nineteenth century, during which period bronchiectasis was well known pathologically, and most of the ætiological features had been discussed and to some extent elucidated by the great pathologists of the nineteenth century. The first clinicopathological knowledge of any note was that of Laennec, who had discussed that important matter in 1819. Virchow and others had shown that the primary change in bronchiectasis was probably in the bronchi, any pulmonary changes being of a secondary nature. Stokes, of Dublin, had laid stress on lack of muscular action of the circular muscle fibres of the bronchi, and also on lack of ciliary action. He had expressed the opinion that pulmonary infections, especially those of childhood, were of considerable ætiological importance. Corrigan, of Dublin, had considered that traction on the bronchi from intrapulmonary fibrosis was a factor of importance. Rokitansky had first clearly differentiated bronchiectatic from tuberculous cavities.

Clinically, early bronchiectatic changes, and even late cases, had been frequently confused with pulmonary tuberculosis, until Sicard and Forestier, in 1921, visualized the internal lumen of the bronchus by means of poppy-seed oil impregnated with 40% iodine solution. Since that time it was possible to delineate accurately the exact localization of the bronchiectatic changes, and to some extent to evaluate their severity. That had really made possible the third era, that of surgical removal of localized diseased portions.

The surgical era had been pioneered by a number of American, English and Continental surgeons, amongst whom Graham, Lilienthal, Sauerbruck, Roberts and others

were prominent. Their technique and results would be discussed more competently by other members at the meeting.

Dr. E. Hughes-Jones said that the surgical position had improved beyond the expectation of a mortality of 64%. With reference to the patients shown that night, he said that, when dealing with children, the medical attendant could afford to wait for years if there was but little sputum. The surgical problem was not one of saving life at that age. The prognosis was still unknown because radical surgical methods had been adopted only recently. The complications, when they occurred between the ages of sixteen and twenty-five years approximately, were so dreadful that the medical attendant regretted that the operation had not been done at an earlier age. Those complications were gross suppuration leading to pyopneumothorax, broncho-pleural fistula and formation of chronic empyema cavities. Surgical treatment had to be carried out, but the patients had to be selected for it carefully. The results of operation were disappointing at times. A boy, apparently in good health, had died of diffuse bronchopneumonia sixty hours after operation. They could afford to wait a long time before operating on the children shown that night, but the decision should not be delayed too long. Churchill, in America, would not operate unless the patients were producing a lot of sputum, were suffering considerably in health, and unless they or their relatives demanded that something of the sort should be attempted. Churchill had stated that he had not seen improvement in bronchiectasis from operations on the nasal sinuses. Surgeons should be careful not to operate on the subjects of bronchiectasis in winter, if respiratory infections were common, or at the height of summer, on account of the loss of fluid from the body brought about by the hot, dry air. The patients should be kept under careful observation, with special attention to the clinical state of the rest of the lungs; and operations should be performed only on suitable patients at the appropriate time. At the operation the anæsthetist had to avoid the least cyanosis, as cyanosis made the prognosis very bad.

Dr. Hughes-Jones thought that at times postural drainage proved unsatisfactory if the patient had to lie on a hard table. He had found it more satisfactory to have the patient lying across a bed with the head down towards the floor and later with the chest over the edge of the bed. The patients were willing to carry out the procedure for longer periods by those means.

Dr. T. G. Millar, in reference to diagnosis, stated that there was considerable dissatisfaction with lipiodol work and that facilities *de luxe* were required. It was of advantage to combine bronchoscopy with the radiological procedure, and, if necessary, to give two or three instillations of iodized oil in order to study the different areas thoroughly.

Dr. Millar then stated that medical practitioners who were not rhinologists held that any sinus disease that might be present had to be eliminated. In their work rhinologists were not satisfied with radical antrum operations or incomplete upper sinus operations for those patients, but pan-sinus surgery was sometimes indicated. Patients under fifteen years of age gave better results as a rule than older ones, though the operations did not cure the bronchiectasis. The patients could, however, be kept at school and out of bed. Though the chest condition would not be cured the major troubles might be cleared up. In conclusion, Dr. Millar expressed the opinion that bronchoscopy as a method of treatment of bronchiectasis was useless.

Dr. Ian Wood showed a series of lantern slides to illustrate the point that bronchiectasis was prone to occur at the site of antecedent atelectasis, and that operative removal of the collapsed portion of the lung at an early age might prevent extension of infection to the rest of the lung. He demonstrated from the slides the various steps of an experiment he had carried out to establish the mode of production of the triangular shadow at the base of the lung. He had obtained two collapsed lungs from a new-born baby who had not survived for more than a few minutes owing to bilateral atelectasis. The baby had been born cyanosed; and it had been established by microscopical examination of prepared sections that the alveoli were collapsed. The bronchus leading to the left lower lobe was ligated and the remainder of the lungs was inflated with oxygen; a triangular shadow, occupying a position behind the heart, was visualized in a skiagram. That shadow had disappeared when the ligature was released. Dr. Wood stressed the importance of collapse of the lower lobes; the collapsed lobes acted as areas of low resistance for the production of bronchiectasis. They usually became infected, and later infected the surrounding lung tissue. The collapse could be either congenital or acquired. Sometimes the collapse was intermittent, and mechanical inflation was occasionally possible.

Dr. Wood remarked that if obvious infection of the collapsed portion was present lobectomy appeared to be indicated. Surgical treatment was also probably wise if there was some infection of the surrounding lung tissue, which might be induced to heal if the "sump" was removed. On that proposition could be founded the advocacy of early lobectomy if infection of the rest of the lung had not advanced too far.

Dr. J. G. Whitaker said that the surgeons at the Children's Hospital did not rush the operation of lobectomy for bronchiectasis, and only two patients had been subjected to the operation there. From what he had seen at operation, however, he was satisfied that the lung affected was carnified and was quite different from the unaffected portion. It appeared to act as a cesspool, and could not be treated medically with any prospect of benefit. He considered that the presence in the skiagram shown by Dr. Ian Wood of the triangular shadow indicative of collapse of the left lower lobe was of considerable significance as an indication for operative treatment. According to the experience of the workers at the Alfred Hospital, by bronchoscopy the lung could be cleared of infected mucus preoperatively, and by that procedure post-operative risks were reduced. The question of anæsthesia was important, and he believed that the use of spinal anæsthesia had been adopted at the Alfred Hospital and held out the greatest hope.

Dr. Russell Howard said that the patient shown by Dr. Wait had unilateral, unilobar bronchiectasis, which was universally recognized as the most suitable type of lobectomy. It had yet to be decided for certain whether lobectomy should ever be performed for purulent bronchiectasis. A decision on that point required a discussion of the results of medical treatment if surgery was refused, and of the operative mortality of lobectomy and the ultimate prognosis if lobectomy was carried out successfully. With reference to the results of medical treatment alone, Dr. Howard quoted from the literature of the past ten years. Chandler, in *The Lancet* of October 27, 1934, had said that the moment the sputum became offensive the prognosis was bad. The amount of pus also appeared to be a factor. There was always a possibility of cerebral abscess, though this in his experience was not common. But the danger of bronchopneumonia was ever present, and also a tendency to progressive myocardial degeneration. Dr. Howard remarked that in face of those facts the prognosis with medical treatment alone was not very good. He went on to state that Chandler had expressed the opinion that lobectomy in suitable cases should be curative and that soon bilateral cases would yield to the skill of the surgeon. Dr. Howard thought that that time had arrived.

He then stated that Roles and Todd, in *The British Medical Journal* of October 7, 1933, had reported the results of medical treatment at the Brompton Hospital over a period of three to six years from diagnosis. Of forty-nine patients twenty-three had died, nine were totally incapacitated, eight were partly incapacitated and only nine were well. They had concluded that patients with infected sputum, whatever the anatomical distribution of the disease, progressed steadily to a fatal issue if they were treated medically.

In a series of cases reported by Leonard Findlay and Stanley Graham in *Archives of Disease in Childhood* in 1931, thirty-two patients had been observed for periods varying up to seven years, and twelve of them had died. In excellent bronchograms the progress of the disease had been followed in most of those who were still alive, and a few in whom the lesion was of short duration had appeared to be cured. Those writers had concluded that

the prognosis in bronchiectasis in childhood was grave, as the condition usually steadily grew worse and terminated fatally. Dr. Howard remarked that, as the children in the series reported by Findlay and Graham were all under the age of fourteen years, the conclusion reached by them was especially applicable to the discussion that night. He went on to say that he had not been able to find any English statistics other than those he had quoted, and that though admittedly the climate in Australia might conduce to less grave results, the mortality rate of almost 50% would need to be reduced very considerably before it approached the better results from surgical treatment which he proposed to discuss later. In addition, the more favourable climate should conduce to better operative results and a lower recurrence rate.

Dr. Howard said that the operative mortality rate abroad had fallen considerably. In the last fifty cases in which he had employed lobectomy for bronchiectasis, Tudor Edwards had had a mortality rate well under 10%. Churchill, in 1937, had reported a series of thirty-eight cases with only one death. O'Brien had also reported a series of fifteen cases with only one death. Dr. Howard remarked that the mortality was quite comparable with that of appendicectomy in acute appendicitis; and the results of medical treatment alone were much worse than would be anticipated in acute appendicitis.

He then discussed the ultimate prognosis after lobectomy, and said that from his experience with Tudor Edwards he could say that most of the patients were well and without recurrence three to four years after lobectomy performed strictly for unilateral and unilobar bronchiectasis. Undoubtedly some patients would develop bronchiectasis elsewhere. They had probably had weakened bronchial walls before operation from spilling of pus from the affected lobe. That was a point in favour of earlier operation rather than a contraindication to it. He had no doubt that some large series of cases would be reported shortly which would support his statement about the low rate of recurrence. From a consideration of the argument he had advanced, Dr. Howard expressed the opinion that there was little to be said against lobectomy as the appropriate treatment for the children shown that night. He thought that the patient shown by Dr. Wait should have the operation performed the following summer, and that the patient shown by Dr. Ian Wood should also be subjected to operation in the hope that ultimately the bilateral disease would be overcome by surgical means and that the patient might join the steadily increasing number of subjects of successful bilateral lobectomy.

Dr. Wait, in reply stressed the necessity for cooperation between physicians and surgeons in the management of bronchiectasis. The condition of the patients had to be followed up carefully over the years. By the earlier use of lipiodol and radiography earlier diagnosis would perhaps make it possible for something of value to be done for them. Of a number he had under observation, in only two patients could the condition be classified as unilateral and unilobar. With reference to the sinusitis which was usually an accompaniment, Dr. Wait thought that cure and not alleviation of symptoms only should be the aim of treatment.

Dr. J. E. Sewell added that the results in England were extremely good when the surgery was done by men who spent nearly all their time at thoracic surgery. Opportunity should be given in Australia to those who were prepared to make it a special study.

(To be continued.)

Obituary.

WILLIAM COLIN MacKENZIE.

William Colin MacKenzie, who died on June 29, 1938, at Melbourne, was born at Kilmore, Victoria, on March 9, distinguished academic career and graduated Bachelor of 1877. He was educated at Kilmore State School, Scotch College and the University of Melbourne. He had a Medicine in 1898. He obtained the degree of Doctor of Medicine in 1901. He will be most remembered by the profession in Australia as the founder and first director of the Institute of Anatomy at Canberra. He presented his own unique and valuable anatomical collection to the Commonwealth. He had been offered a large sum of money by an American institution for this collection; but he would not consent to its removal from Australia. His studies in comparative anatomy and in the reeducation of paralysed muscles received world-wide recognition. In 1929 he received the honour of knighthood.

The nation is the poorer for his passing. The sympathy of the medical profession in Australia is extended to Lady MacKenzie in her bereavement.

We are indebted to Dr. C. V. MacKay for the following account of William Colin MacKenzie's career.

The death of Sir William Colin MacKenzie, M.D. (Melbourne), F.R.C.S. (Edinburgh), F.R.S. (Edinburgh), the founder and first director of the Australian Institute of Anatomy at Canberra, means a definite loss to the medical profession of Australia.

He had been in failing health for a considerable time. Like several other well-known anatomists, he paid the penalty of a comparatively early death for too tense an absorption in a particular branch of human study. Increased blood pressure, with its concomitant effect on the cerebral arterial system, resulted eventually in a major hæmorrhage.

William Colin MacKenzie belonged to that very small band of Australian medical men whose work and writings are known beyond the confines of our island continent.

It can hardly be said that as yet the influence of Australian thought on medical science in the world has been profound. Until comparatively recently, as might only be expected, contributions to medical literature of an original character have not often been made by native-born Australians. Our resources in this respect have always been depleted by the gravitational pull on our most brilliant minds of the medical centres in the Old World. A striking example of this character was the loss to Australia at an early age of the world-famous anatomist Sir Grafton Elliot Smith. Professor John Hunter, of Sydney, unfortunately lost his life during his visit to America and England, and Professor Woollard, among many others, has remained in London.

Although William Colin MacKenzie visited Europe on four occasions, and America twice, he always returned with his devotion to his homeland unimpaired. His loyalty to Australia, accompanied by his intense desire for the furtherance of everything which would tend to advance Australia and add to its scientific prestige, was almost a passion with him.

William Colin MacKenzie had a most unusual professional career. He graduated as a comparative anatomist from the ranks of general practitioners of medicine.

He was born on March 9, 1877, at Kilmore, in Victoria, a small town on the main north-eastern road from Melbourne to Sydney. The youngest of six children, he was the third of four sons to be educated for the medical profession. From the State school at Kilmore he won a three years' residential scholarship at Scotch College, Melbourne, and matriculated to the University of Melbourne with honours in Greek.

During his medical course, which he completed within a few months of his twenty-first birthday, he obtained first-class honours in medicine and in surgery, and became one of the six resident medical officers at the Melbourne Hospital.

A further two years as senior resident medical officer at the Children's Hospital, Carlton, fitted him to commence general medical practice at North Melbourne, near the gates of the university, in 1901. Soon afterwards he was appointed demonstrator of anatomy at the medical school of the University of Melbourne, and subsequently lecturer in applied anatomy. MacKenzie's interest in work with children was increased by his early appoint-

ment as clinical assistant to out-patients at the Melbourne Children's Hospital. One of his earliest studies was upon syphilitic dactylitis, and for two years before Schaudin's momentous discovery of the spirochæte of syphilis, he urged the officers of the bacteriological department of the University of Melbourne to collaborate with him in a search for the causal organism.

In 1904 MacKenzie paid his first visit to Europe. This was destined to have a very great influence on his professional work and future career. On his way to Edinburgh, where he obtained by examination his fellowship of the Royal College of Surgeons, he stopped at Heidelberg, in Germany, to work with Professor Vulpius, the well-known orthopædic surgeon.

After leaving Edinburgh, where he had heard of the splendid work being done by Robert Jones at Liverpool, he proceeded to investigate the practice of the conservative school of orthopædics founded by the famous Hugh Owen Thomas in that city.

Returning to Melbourne invigorated by the example of the new operative work on tendons being performed by Vulpius, and greatly influenced by the principles of muscle rest and muscle recovery then being enunciated by Robert Jones, he found the Children's Hospital faced with a severe epidemic of infantile paralysis. The knowledge of the various new splints then being used at Liverpool, and of the basic ideas which govern their use in the treatment of muscle pareses, was at that period possessed by comparatively few medical men in Australia.

The treatment of the recent and remote effects of the scourge of infantile paralysis on children was during the ten years preceding the Great War the master idea in MacKenzie's mind. He was an innovator and not a slavish copyist of other men's ideas. Although greatly influenced by Thomas, Vulpius and Robert Jones, he was continually attempting to improve upon their methods.

In the surgery of old cases of poliomyelitis his aim was always to relieve the weak muscle groups by tenotomy of the tendons of the overacting muscles, followed by adequate rest and accompanied by reeducation of the weak groups.

In early treatment, before the onset of crippling deformities, he for some time followed the lead of Robert Jones by splinting the overstretched muscles in the position of relaxation. Gradually he went further than this, and, not content to wait until simple relaxation produced recovery, he evolved his theories relating to the fascinating subject of muscle reeducation. These slowly took shape until they were ultimately embodied in his text-book "The Action of Muscles, including Muscle-Rest and Muscle Re-Education".

Although commonplace today, MacKenzie's original conceptions were revolutionary. He was the first to use the special term "muscle reeducation" and to stress the great importance of the action of gravity in all educational attempts to regain muscle function after paresis of any nature. The minimal load, the zero position, the smooth powdered surface of board or cardboard for reeducational muscle work must always be associated with MacKenzie's name. Sir Arthur Keith, in his remarkable book "Menders of the Maimed", published in 1919, gave MacKenzie full credit for this definite advance in medical treatment.

During the ten years previous to 1914 MacKenzie had gradually found time to investigate the special anatomical problems associated with native fauna of Australia, which were afterwards to play a predominant part in his life.

It was during a dissection of the deltoid region in the koala that he became especially interested in the functional application of such an unusually well-developed muscle. Function and structure as the two essential correlations of life were constantly in MacKenzie's mind and inspired his work. Watching the koala in his native environment, he carefully observed its power of raising the hand and arm above the head. For a considerable time he had been much perturbed by the bad results following infantile paralysis of the upper arm. The almost absent wasted deltoid with the dropped head of the humerus and dangling arm became a nightmare to him. Out of these observations and anxieties was born the deltoid or upper arm abduction splint for use in all cases of upper arm paresis. MacKenzie undoubtedly was the inventor of this splint. His type of abduction splint was featured in his writings in 1908, and the splint itself was sent to an orthopædic congress and exhibition in Bologna, Italy, in that year. I have been unable to find any reference to a splint of any kind for this purpose before MacKenzie's publications.

There can be no question that this was a very great advance in treatment, comparable with the Thomas hip splint and the knee caliper.

Although numerous modifications have been introduced at various times, the essential principle of gravitational rest to the deltoid and shoulder girdle muscles remains the same. MacKenzie's original type of splint was again illustrated in an article published in *The Australian Medical Journal* on March 16, 1912, entitled "Some Anatomical Considerations of Joint Fixation". A subsequent article, "The Principles Governing the Early Treatment of Infantile Paralysis", published in *The British Medical Journal* of February 24, 1917, gave a clear illustration and full instructions for use.

A recent visit to Dr. Böhler's special insurance fracture clinic in Vienna showed that practically every case of arm injury was treated on an abduction splint to obtain rest for the whole arm. There can be no question that the discovery of the application of the deltoid or abduction type of splint was a theoretical and practical advance in treatment.

As his work on the after-results of infantile paralysis developed, MacKenzie retired from general practice. This was soon after his return from Europe. He then specialized in orthopædic surgery in Collins Street, Melbourne, devoting any spare time to dissections of the Australian fauna.

The present writer has vivid recollections of the two or three years prior to the Great War, when every evening and every week-end was given up to the unravelling of the anatomical details of the platypus, koala, wombat *et cetera*. A faithful old copy of Richard Owen's "Comparative Anatomy of the Vertebrates" was our inseparable companion in this work. At the outbreak of war quite a large collection of dissections had been accumulated. In the early part of 1915 MacKenzie proceeded to England to assist Sir Arthur Keith in the cataloguing of war specimens from France *et cetera*, and to continue his anatomical researches. In 1917 he again met Sir Robert Jones, and opened for him a muscle reeducation department at the special orthopædic military hospital at Shepherd's Bush, London. It was during this year that he wrote, with the assistance of his cousin, Major Charles MacKay, R.A.M.C., "The Action of Muscles", which embodied all his thought and experience on this subject over many years. It was the first text-book to use the muscular systems of the native fauna of Australia as illustrations to assist in the correction of muscle and joint disabilities. Comparative anatomy with functional adaptations was included at every opportunity.

This book, published in 1918, when its author was on his way back to Australia, was an immediate success. The first edition was reprinted in 1919 and a second edition printed in 1930 is now nearly exhausted.

After the Great War MacKenzie gradually devoted himself more and more to comparative anatomy, and his collection of Australian faunal specimens eventually became too great for any private individual. Negotiations were opened with the Commonwealth Government to acquire the collection as a gift to the nation. These were successful, and culminated in 1924 in an Act of Parliament by which the Government of Australia undertook to house and maintain the dissections *et cetera* at Canberra, and to appoint the donor first director with the title of Professor of Comparative Anatomy.

Subsequently William Colin MacKenzie received the honour of knighthood. In 1930 the collections were transferred to Canberra, and have since been housed, with many additional gifts, in the two spacious well-lit museums attached to the Australian Institute of Anatomy. This building, which occupies a commanding position in the new Federal capital, is an imposing and beautiful example of modern architecture. Both within and without the

structure replicas of the Australian fauna are incorporated wherever possible. The female platypus is enshrined in the glass roof of the large entrance hall, and koala heads grace the surrounding walls of the central grassed courtyard.

The national feeling of our race is well exemplified in the capital city by this glorification of our unique animals. It augurs well for the future scientific spirit and cultural atmosphere that such an academic science as comparative anatomy should be so well housed.

The anatomical museums, which are open to the public without charge, from 8 a.m. to 5 p.m., are very well patronized. They will be a lasting memorial to the vision and genius of one who loved Australia and its wonderful scientific heritage with all his heart and mind.

The future of the institute from an anatomical point of view, based on MacKenzie's opinions and policy, is uncertain. The recent appointment, as director, of a senior medical officer of the Commonwealth Department of Health, who is an expert in the science of nutrition, augurs clearly an experimental physiological rather than an anatomical basis for future development.

MacKenzie derived much of his force of character, his desire for knowledge and his love of educational facilities from his mother. This lady was the eldest daughter of Charles MacKay, a pioneer schoolmaster from Bonar Bridge, in the far north of Scotland, who came to Australia immediately following the church disruption troubles in 1848. MacKenzie was devoted to his mother's memory and to her ideals. In her honour he founded "The Anne MacKenzie Annual Oration" at the Institute of Anatomy, with an endowment of £1,000. This great mark of filial respect and affection, which, although copied from Britain, was an innovation in Australia, has so far produced several notable orations.

During a very active medical life MacKenzie published many articles, dealing mainly with infantile paralysis, in *The Intercolonial Medical Journal*, The Medical Journal of Australia, *The Lancet* and *The British Medical Journal*. Later on, as he turned from practical medical problems to those of comparative anatomy, some of his works were published in *The Journal of Anatomy*; but most of them were publications of the institute itself. Some early writings were published privately. The following is a brief list of some of his better-known writings:

"The Action of Muscles, including Muscle-Rest and Muscle Re-Education."

"The Larynx in Australian Monotremes and Marsupials."

"The Shape and Peritoneal Relationships of the Spleen in Monotremes and Marsupials."

"The Genito-Urinary System in Monotremes and Marsupials."

"The Liver, Spleen, Pancreas, Peritoneal Relations and Biliary System in Monotremes and Marsupials."

"The Glandular System in Monotremes and Marsupials."

"Military Orthopædic Hospitals", *The British Medical Journal*, May, 1917 (a special War Office publication).

"Intellectual Development and the Erect Posture", 1924.

"The Treatment of Infantile Paralysis: A Study in Biology", *The British Medical Journal*, 1915.

"The Human Erect Posture", The Medical Journal of Australia, 1926.

"Australian Fauna and Medical Science", *Papers and Proceedings of the Royal Society of Tasmania*, 1926.

"Treatment of Extreme Infantile Paralysis of the Lower Limb in Children", *The Intercolonial Medical Journal of Australasia*, 1906.

"The Principles Governing the Early Treatment of Infantile Paralysis", *The British Medical Journal*, 1917.

"A Contribution to the Biology of the Vermiform Appendix", *The Lancet*, 1916.

"Further Studies on the Muscular Factor in Infantile Paralysis", *The Australian Medical Journal*, 1911.

"Studies on the Biology of the Thymus Gland", *The Australian Medical Journal*, 1914.

William Colin MacKenzie was married in 1928 to Dr. Winifred Smith, M.B., B.S., of Melbourne. There are no children.

Dr. William Crowther writes:

In the years between 1906 and 1910, as medical students at the University of Melbourne, we were fortunate in having as our professors such outstanding men as Baldwin Spencer, Masson, Lyle and Allen, all of whom were subsequently knighted for their work in science, and three already with the coveted "F.R.S." after their names. Among the tutors and demonstrators, younger men and recently qualified, I best remember T. P. Dunhill and Colin MacKenzie, who eventually were to rank with the men already named.

MacKenzie as demonstrator in the School of Anatomy was a distinctive figure. Painstaking, careful and very friendly to his students, he was invaluable to his chief. The latter, R. J. A. Berry, had just taken over the Chair of Anatomy, and to his demonstrators fell much of the work of meeting his insatiable demands for frozen sections of the human body in all planes, and innumerable other preparations for permanent mounting in the museum. Such close anatomical study in association with his clinical work at the Children's Hospital and a very large private practice, prepared the ground for MacKenzie's original conceptions in the treatment of infantile paralysis. His method of splinting damaged muscle groups until function was restored, wholly or in part, attracted much attention, and his results were outstanding. From this period his mind largely concerned itself with the action of muscles in their relation to posture. His experiments with our native fauna inevitably followed.

Of his war work I have no personal knowledge; we do know that his services were eagerly accepted by the Imperial Government.

In 1928, as President of the Zoological Section of the Australian and New Zealand Association for the Advancement of Science, his address on "The Importance of Zoology to Medical Science" was of great interest.

For me a visit to Melbourne was not complete without going to his wonderful laboratory and collections at St. Kilda Road, when he would show any new material and explain his recent research. Fortunately for science, the Australian Government acquired this collection (his life's work) and built at Canberra the splendid Institute of Anatomy to contain it. Here, as director, Sir Colin spent the remainder of his working life.

His was the fate of the original thinker, not only to stimulate thought, but at times far-reaching controversy. His services to Australia will become of more and more importance as research workers of the future make use of the material that he alone made available to them. His friends will always retain their memory of his distinguished person, with its gentle and kindly soul.

Dr. J. H. L. Cumpston writes:

Many men have dreams, few have the gift of formulating practical dreams, and still fewer have the ability and determination to carry their visions to established reality.

Before the War (about 1911) Colin MacKenzie gradually approached and defined a concept which may well be expressed in his own words:

> In the consideration of any diseased tissue of the human body, such as cancer, a comparison must be made with the condition in health—the abnormal must be compared with the normal. Thus arises the question, what is normal mammalian tissue? Recognizing the effects, over centuries, of alcohol, syphilis and other poisons on the human race, one would be loath to regard tissues from an individual dying from misadventure or natural causes as typically mammalian, and similarly with animals commonly used for experimentation, such as dogs, rabbits and guinea-pigs, owing to the modification of domestication. It is to the primitive mammals of Australia and Tasmania, unaffected by syphilis, alcohol or domestication, that have lived in a natural environment for millions of years, that we must look for normal tissue. In the

case, *e.g.*, of the ductless glands, the platypus (*Ornithorhynchus anatinus*) offers a remarkable standard for human comparison. Thus the parathyroids are constant, and easily found at the junction of larynx and trachea; Cowper's glands, rarely seen by the medical student, are highly developed; the thymus is retained in the adult; the three ductless glands not so far discovered in us can be demonstrated, *vis.*, parathymus, scapular and sex glands.

His enthusiasm for this idea was not merely theoretical; for at his own expense he engaged a staff and began the study of Australian animals and the collection of anatomical dissections and histological sections. This collection he ultimately offered to the Commonwealth Government, which accepted it and established the National Museum of Australian Zoology, passing an enabling statute through Parliament in 1924 and erecting the existing building at Canberra in 1929. It was at this stage that I first came in contact with Colin MacKenzie's dream and his work. His vision had by then become somewhat more extensive, as he realized how valuable a national asset such an institute at Canberra could be. When giving evidence, in 1928, before the Public Works Committee, Colin MacKenzie expressed this wider vision in these terms:

> The institute is now under the Department of Health, and it constitutes an important adjunct to the department's activities. The institute will become one of the most important in the world, occupying in Australia a place in the scientific world like that of the Hunterian Museum in connexion with the College of Surgeons in England, which is the largest museum of its kind in the world. It was established 200 years ago. During the Great War the whole of the medical activities in Great Britain were concentrated in that museum. If any problem arose in relation to medical research it was referred to the authorities of that institution for determination. It is an accepted fact that anatomy is the basis of all medical and surgical problems. It will be seen, therefore, that the Department of Health now has a most important weapon under its control as regards the general health of the community.
>
> The Institute of Anatomy will be in a sense linked up with the proposed Institute of Public Health in Sydney, because both institutions will be concerned with the general health of the people.

This enlarged conception was validated by the statute of 1931, which changed the title of the institute to the "Australian Institute of Anatomy".

In 1929 Colin MacKenzie's work was recognized by His Majesty with the honour of knighthood.

In my discussions with Sir Colin in the early days of our official association I was impressed with two essential aspects: first, his great admiration for Peter Bennie, especially in relation to Bennie's insistence on the proper growth and development of the child; and, second, his almost passionate devotion to the concept that the ultimate objective of the study of the primitive animals was greater knowledge of the principles underlying the correct development of the muscular and skeletal systems of man, both in health and disease. His study of "erect posture", about which sometimes we used to make gentle fun, had a real underlying significance.

Sir Colin developed the new institute along the lines of his vision, until it has now become known to the public throughout Australia. His devotion to his vision was intense and unwavering, and, realizing that the limitation of the work both in time and extent could not now be defined, he was content to lay the foundations solidly and well. It was most unfortunate that the period of economic depression came at the time when he was really beginning to develop the possibilities of the institute; but even then he met the situation to some extent from his private funds.

The department is continuing, as far as this is possible in the absence of its author, the vision as we understand it. In linking the continued study of the Australian fauna with the study of the development of the growing child, we shall be taking the first of many successive steps towards the realization of Colin MacKenzie's original and splendid dream.

Dr. Edmund Vance writes:

It was a great privilege to have known well so remarkable a man as Colin MacKenzie.

When Kipling wrote:

> "The men bulk big on the old trail, our own trail, the out trail.
> They're God's own guides on the Long Trail—the trail that is always new",

one feels he had in view our profession and such men as MacKenzie.

As an orthopædic consultant, in which role I met him first, in immediate post-War years, he was sagacious, sincere, unperturbed and polished—a comfort both to patient and practitioner. Had he remained at this post he was assured of professional fame and financial fortune. But he chose another course.

In the capacity of master to disciple he gave much of his extensive knowledge and good sound counsel. As a disciple, one learnt of the sources of his mastership. It was research work in pure science.

As a comparative anatomist he was gifted with insight; he was devoted passionately to his work, at which he was capable of intense application. If "he who lives well is the best preacher", as Cervantes says, then this master preached well the gospel of ardent, assiduous and prolonged toil, by his life.

In taking the fauna of Australia as a research field, hitherto unexplored, for study, he was cultivating that part of comparative anatomy which was unavailable to the great John Hunter in his studies more than a century ago. The Australian mammals, isolated geographically, through long ages, from those of other lands, greatly differed in their evolutionary development from them; and to the thinker who was now dealing with the subject this field proved rich in suggestive themes and observations which threw light on problems of human form and function.

He wrote and published "The Action of Muscles", wherein, and with the aid of his research, he was able well to trace the evolutionary histories of most of the main muscles and to offer great help to the surgeon engaged in muscle reeducation. His dissections engrossed him. His museum became famous.

One saw him then begin another stage of his life. Devoted as he was to Australia, and desirous of giving Australian medical scholarship the advantage of a sound foundation on comparative anatomy, he set himself, fourteen years ago, the gigantic task of bringing into being the Institute of Anatomy at Canberra. Thus engaged, one saw a new facet of his character, the executive, in which his diplomacy was only equalled by his tenacity of purpose. At last, after much wearying effort, the day arrived when at an excited, happy and historical luncheon he was able to tell me that the grant for the building of the institute, which was to be the nucleus of the future University of Canberra, had been passed.

It was at Canberra, which became for me, what one feels it should more and more be for more and more of us, a medical Mecca, that one came to known Colin MacKenzie, now bearing a title of his worth from the King, as a friend. It was in this role that he was at his best—sympathetic, helpful, humorous, and quite charming. Cherish his memory.

Of John Hunter and his dissections a contemporary is quoted to have said, "His museum is as much use as so many pigs' pettitoes", and yet today John Hunter's museum is a root source of knowledge and inspiration for the medical man all over the world, and for the surgeons of Great Britain in particular, and John Hunter's name stands at the very peak of the great names in the history of British medicine. Perhaps in our treatment of Colin MacKenzie we too, his contemporaries, have been somewhat isolated, apathetic and perverse. But what will be the

judgement of posterity? One feels sure that, as time advances, more things will appear in his work and in the records of it, which will enable the world to judge better of the character and quality of this remarkable man. Gradually, opinion will form which will accord him first place amongst our medical workers, and his fame will increase with the years to come. Australian medical men of the future will betake themselves to the institute at Canberra to avail themselves of the material provided for the study of their problems, and for mental refreshment, and for accommodation provided for research work, as was Colin MacKenzie's dream.

Sir Alan Newton writes:

I first met Sir Colin MacKenzie thirty-two years ago, when I was a student in the dissecting room of the Department of Anatomy in the University of Melbourne. Even at that stage of his career, when he was engaged in a busy practice, he showed his love for the subject of anatomy by finding time to serve as an honorary demonstrator in this department. He was not content to instruct us merely in the relationships of anatomical structures in the human body, but strove also to inspire us to correlate human and comparative anatomy and, more particularly, to read the works of John Hunter. He maintained a keen interest throughout his life in the welfare of all those who had been his students.

Others will write of his distinguished career, of the days when he established a private anatomical museum in Melbourne, of his work on the treatment of poliomyelitis, and of the foundation and development of the Institute of Anatomy at Canberra. In any event these are matters of common knowledge in this country. When I heard of his death, my memory recalled early student days, and I feel impelled to express my sincere gratitude, which is shared, I know, by a great number of my contemporaries, not only for his unselfish devotion throughout his life to the promotion of anatomical knowledge, but, more particularly, for his personal help and inspiration at that time.

JOHN STODDART BARR.

We regret to announce the death of Dr. John Stoddart Barr, which occurred on September 15, 1938, at Troon, Scotland.

HAROLD WALTER BROADBENT.

We regret to announce the death of Dr. Harold Walter Broadbent, which occurred on September 17, 1938, at Adelaide, South Australia.

Correspondence.

THE OPEN CASE OF PULMONARY TUBERCULOSIS IN RELATION TO CLOSE CONTACTS IN FAMILIES AND THEIR ENVIRONMENTS.

Sir: From a general survey comprising all available clinical data at the Westwood Sanatorium, comprising 1,025 cases of pulmonary tuberculosis, it was elicited that 190 patients admitted were due to close contact with open cases, that is, 18·6%.

Included in this survey there were 20 cases of "conjugal phthisis", and 349 children had been in close contact with their tuberculous parents.

This is a matter of paramount importance, not only from the public health point of view, but also as it affects the individual and the State, both socially and economically.

In metropolitan areas and larger towns where there are tuberculosis clinics, open cases can be taken in hand, and their immediate contacts. On the other hand, the difficulty arises of exercising supervision of open cases in country districts, and every endeavour should be made by the public health departments of the various States to overcome this.

Transfer of children from tuberculous to healthy homes (the Grancher system) is in vogue in France. In 1918 there were 600 homes, and in 1936 this had increased to 8,000. In New South Wales there are preventoria for children, and this system could possibly be adopted in other States.

Conclusions.

1. The principal aim should be to regard the patient as a "family unit", and all the immediate contacts should undergo the following investigation: a clinical examination, X ray, sputum and skin test.
2. Whatever the source of infection, this should be systematically investigated.
3. Patients should be advised under the following headings: (*a*) with regard to themselves and their children; (*b*) treatment, domiciliary or institutional; (*c*) as to any future marriage contemplated; (*d*) greatest age of susceptibility.

Yours, etc.,

J. H. Blackburn,
Medical Superintendent,
Westwood Sanatorium.

Queensland,
September 3, 1938.

NATIONAL HEALTH INSURANCE.

Sir: Why on earth will not the profession as a body definitely refuse to have anything to do with the national health insurance? It is fundamentally wrong and cannot be made right at any price. People having to pay for something they are not getting at the time, and later doctors giving services (often unnecessary) when not receiving reward at the time, is a travesty of the principle of give and take.

This lodge system has always been nothing but annoyance to all concerned from start to finish. It is a wrong attitude between doctor and patient. A private patient comes to the doctor with faith, trust and a sometime desire to pay for valued services. The lodge patient or insuree comes only with the "might as well get as much as we can out of him, it's our due" attitude.

Do doctors want interference by outsiders in their own business all the time, official naggings and multiple letter writings? How reminiscent of lodge practice it will be, thinking out arguments to baffle each back-biting insuree who reports one, mostly because he has an inferiority complex, because he is not paying at the time and therefore thinks he is not "getting a fair deal".

"Bureaucrazy" is the word that the system means. Dr. R. O. Williams (see The Medical Journal of Australia, August 13, 1938, page 257) and Dr. E. Brettingham Moore (see The Medical Journal of Australia, August 27, 1938, page 357) and others have correctly stated the case. Admittedly it is a very clever idea on the politicians' part to use our profession to finance all sorts of schemes, reduce unemployment *et cetera*. I do not blame them a bit, if we are fools enough to take it. Officially resist such measure *in toto* is the correct British Medical Association attitude. To say that "It can't be done because some members would 'scab' " is an absurd bogy argument.

As for north Queensland, I do not know of any practitioner who wants national health insurance, and it is totally repulsive to most. Concerning the practitioners in the city of Townsville, I would vouch that none would turn infidel on such a stand by the profession. And what would it matter if some, somewhere, did? The slave-driving national health insurance system would give them the hiding they deserve.

Try a stand and see. I, for one, do not believe there would be the described caving in, in this instance. Would

the British Medical Association please take a referendum vote from all its members on the subject and just see their attitude?

Yours, etc.,
A. G. BENNETT, M.B., Ch.M.

Townsville,
Queensland,
September 5, 1938.

SIR: Dr. Michael Kelly, of Bunbury, Western Australia, has voiced the opinion of a very large section of the British Medical Association practitioners when he advocates the complete rejection of the Government scheme as at present outlined.

Doctors will be individually competent to accept or reject the contract. If an overwhelming number are in favour of rejection, as may be disclosed by a "plebiscite", then it is possible that we practitioners will still retain our liberty and status. There is no doubt that we have the power to do this, and in the opinion of myself and local colleagues it would be sheer folly not to exercise it.

Amongst the many objections put forward to the medical part of the scheme I have not read anything relative to country calls in Queensland. In this State no charge whatever is made for the use of the ambulance. The result of this free service is that in our district (200 miles by 100 miles) 99% of the medical and surgical calls go to the ambulance. It is a rarity for doctors to be called any distance into the country by either private or lodge patients.

To deal with these calls we have a staff of full-time, hard-working ambulance bearers as numerous as the doctors themselves (eleven in all). For the past year no fewer than 7,390 transports to doctors and hospitals were made, involving a car mileage of over 120,000 (*vide* annual report, Mackay Ambulance).

Imagine what the position would be under national health insurance. The doctor could be obtained at no cost, and it would be considered by the patients that they had a right to call the doctor, as he would be paid for it. Were even 30% to 40% of calls diverted to practitioners it is doubtful if they would be physically capable of the increased work and time involved. Certainly there would be no leisure.

Were the capitation fee double the proposed amount there must be many who would prefer the comparative freedom of their present condition. We object to the conditions of service far more than the details of the capitation fee.

Surely a "plebiscite" can be organized by the appropriate authority whilst yet there is time.

Yours, etc.,
ERNEST CHENOWETH, M.D.

Mackay,
Queensland,
September 14, 1938.

SIR: As I read the flood of reports and letters concerning national health insurance, I find a singular lack of emphasis, even in our evidence before the Royal Commission, on what is, surely, the most important point to be considered in fixing a capitation rate.

If one may judge by statements made by its chairman, the Royal Commission proposes to estimate the income that we, as a profession, will lose as a result of the health insurance act, and to give us back the same, or perhaps a smaller amount, through the capitation rate. That is a reasonable procedure provided we continue to do the same amount of work.

My present practice is a comfortably busy one in a one-man town with a practically closed community. With a capitation rate of eleven to fourteen shillings plus the proposed mileage rate it would continue to bring in about the same income as at present. But here is the important point. If national health insurance covered the whole community (which I suggest merely to simplify the argument) there would be a comfortable amount of work for two practitioners—with an income for one!

That the amount of work would be at least doubled, I am quite confident, having had considerable experience of contract practice both in England and Australia. The Bingara practice is again a good illustration, being, as I mentioned, in a closed community (for geographical reasons). I know the number of potential patients in the private and in the contract classes, and I know that the members of the contract class visit me about three times as often per head as those of the private class. Allowing for an average visit of two-thirds the duration (it is certainly not a smaller fraction), it is clear that those people who change from the private to the contract category will, as soon as they get used to the idea, double the amount of work they occasion us. I quite believe that this is a good thing for the community, provided the standard of medical service remains unchanged. But surely it is plain to all that it is impossible to double the amount of our work without either tragically lowering its efficiency or doubling our numbers. The latter, more desirable alternative, would occur by natural processes in the course of time, provided there were no decrease in the inducement to enter our profession; but I am quite convinced that there must be such decrease unless the Government doubles the proposed capitation rate.

The argument is similar and the conclusion identical for the initial transfer of about one-quarter of the population (instead of, as above, practically all of it) from the private to the contract category. Any comparison with the effect of the introduction of health insurance to Britain is quite inadmissible because of the quite incomparable conditions preceding the insurance acts in the two countries.

If the above argument is sound, it behoves us all to insist upon it publicly. Much of the publicity we receive at present seems to me to show us quibbling over pence and details and thereby, I fear, often lessening the respect in which we are held by the community. Let us rather demand a capitation rate of 25s., or else refuse the proposal in entirety. In doing so let us admit that our action is to our own advantage, but also firmly insist that it is equally to the advantage of the community. Then, I believe, we will deservedly retain the community's respect.

Yours, etc.,
T. DUDLEY HAGGER.

Bingara,
New South Wales,
September 12, 1938.

THE PSYCHOLOGICAL APPROACH.

SIR: Your recent leading article on "The Psychological Approach" points out that "a large part of general medical practice consists of illness which . . . is psychologically conditioned" and that the average physician is inclined to ignore this factor.

Could not your journal help us here by giving full and exact working details of the methods of investigation, diagnosis and treatment in use at such a clinic as the Tavistock Clinic in London?

Writers on the psycho-neuroses are as diverse in point of view as they are fluent, and the non-specialist is hard put to find actual working methods which have in fact been well tried with good results.

Yours, etc.,
R. N. BEAZLEY.

1627, Pacific Highway,
Wahroonga,
September 12, 1938.

IRREDUCIBLE PROLAPSE OF THE UTERUS THROUGH A RING PESSARY.

SIR: A woman school-teacher, in middle life, who had had a complete prolapse of the uterus for years, found, after a long day's standing, that the prolapse was down,

with the accompaniment of considerable pain. Attempts to return the organ were futile. She discovered that the ring pessary she was wearing was down also, and that the uterus had come through the ring.

When seen, the whole organ was prolapsed and the ring constricted a very blue, œdematous mass, swollen to nearly the size of a child's head at term. The ring was lying in a deep groove at the utero-vaginal junction, which was just inside or outside the vulva, according as traction was exerted or not. Reduction through the ring was quite impossible. Household tools requisitioned were all inadequate. They included the handle of a table-spoon to protect the tissues during attempts to cut or break the ring, a safety-razor blade to shave off the rubber coverings of the ring and expose the spring, a small triangular file, a hacksaw blade, and the selection of pliers brought in from the tool-shed by optimistic relatives. They all, *privatim ac seriatim*, failed to relieve her of the strangulating relic of mediæval practice. Later, on the operating table, under a good light and outside the protection of a flexible copper retractor, worked in between the pessary and the uterus, the seven layers of spring steel that comprised the core of the pessary were prized apart, one after the other, with a Lane's screwdriver, and snapped in turn with a small pair of wire-twisting pliers.

Having sweated over this job in a distant farmhouse, by the light of a kerosene lamp, before packing the unfortunate woman into my car *en route* for hospital, I wondered somewhat less than formerly whether my refusal for years past to use these unhygienic and dangerous supports in any circumstances were pure prejudice or sound surgery. Too many cases of trouble caused by rings had led me to the conclusion that a woman who refuses operation for prolapse is probably better off with her uterine hernia than with a ring in her "innards" lulling her into a state of false security. And my latest experience makes me feel that the people who insert them should all be condemned (without wishing them any particular harm) to get one out of the snug groove it has made for itself behind a strangulated uterus. They would not find text-book surgery very helpful in planning their method of approach.

I believe it was the fashion about sixty (or is it seventy?) years ago to regard the internal anatomy of the comparatively early Victorian female as so fragile and ill designed as to require the support of rubber, steel and vulcanite, and an X ray picture of a drawing-room full of ladies of the period would have looked, at hip level, something like a bunch of rings in a Chinese puzzle or a juggler's outfit in action. The idea was bad and the remedy for the idea radically unsound. But there would seem to be a revival of this internal fashion, so that one finds gadgets of this nature being used again in women of all ages and conditions. Surely not a revival of the fainting female and the pelvic complex? When one finds, as I recently did, a dear old lady of over eighty wearing a ring pessary of about eight inches diameter for an alleged retroversion, intensely uncomfortable from incontinence and painful micturition, with a blood-stained and malodorous discharge, and actually suffering from a urethral caruncle and vaginal ulceration from the pressure of the mammoth pessary, it makes one wonder.

For those who use a ring pessary as a support in uterine collapse, I would suggest that they cut one across and reflect on its construction. In the trial operation on the affair the following tools should be regarded as unsporting: hammer, cold chisel and anvil, vice and all blacksmithing tools. The use of heat also, to soften the steel, is obviously to be excluded as unsuitable to possible conditions when this form of underwear is being worn. Apart from these, almost anything may be tried. I would also suggest that, having conducted this experiment, Mr. Punch's succinct advice be remembered when about to prescribe the apparatus.

Yours, etc.,

R. M. Crookston.

Camden,
New South Wales,
September 11, 1938.

Post-Graduate Work.

WEEK-END COURSE AT PARRAMATTA.

The New South Wales Post-Graduate Committee in Medicine announces that, in conjunction with the Central Western Medical Association, it will hold a week-end course at Parramatta on Saturday, October 22, and Sunday, October 23, 1938. The programme is as follows:

Saturday, October 22, 1938.

2.30 p.m. to 3.30 p.m.—"Diagnosis and Treatment of Post-Menopausal Hæmorrhage", Dr. R. I. Furber.
3.30 p.m. to 4 p.m.—Afternoon tea.
4 p.m. to 5 p.m.—"Diagnosis and Treatment (including Standard and Recent Methods Abroad) of all the Common Skin Diseases", Dr. J. C. Belisario.

Sunday, October 23, 1938.

10 a.m. to 11 a.m.—"Points of Importance in Gynæcological Operations", Dr. R. I. Furber.
11 a.m. to 11.30 a.m.—Morning tea.
11.30 a.m. to 12.30 p.m.—"Diagnosis and Treatment (including Standard and Recent Methods Abroad) of all the Common Skin Diseases", Dr. J. C. Belisario.
2 p.m. to 3 p.m.—"The Present Situation with Regard to Treatment of Cancer of Female Genitalia", Dr. R. I. Furber.
3 p.m. to 4 p.m.—"Diagnosis and Treatment (including Standard and Recent Methods Abroad) of all the Common Skin Diseases", Dr. J. C. Belisario.
4 p.m. to 4.30 p.m.—Afternoon tea.

The fee for the course will be one guinea. Those intending to be present are requested to notify Dr. K. S. Macarthur Brown, "Brislington", 12, George Street, Parramatta, as soon as possible.

COURSE AT SYDNEY IN CLINICAL LABORATORY METHODS.

The New South Wales Post-Graduate Committee in Medicine announces that a course of instruction in clinical laboratory methods will be given at Sydney from Monday, October 24, to Friday, October 28, 1938. The course, which is designed especially for general practitioners, will occupy the hours of 10 a.m. to 1 p.m. and 2 p.m. to 4 p.m. on each of these five days. For the most part it will take the form of tests and determinations that general practitioners could carry out themselves if necessary. In addition, if time allows, demonstrations of more difficult tests and methods of estimation will be given.

Practical Work.

The actual work done by those attending the course will, to some extent, be determined by the material that offers, but will probably include the following:

1. *Hæmatology*:
   (a) Blood counts: Methods of collection, staining and counting, including counting of platelets and reticulocytes, and estimation of hæmoglobin value.
   (b) Estimation of coagulation time and bleeding time.
   (c) Calculation of the red cell sedimentation rate.
   (d) Blood grouping.
2. *Bacteriology*:
   (a) Collection of specimens for bacteriological examination.
   (b) Routine methods of staining and identification of organisms from direct smears.
   (c) Examination of hair *et cetera* for fungus.
   (d) Examination for *Trichomonas vaginalis*.
3. *Serology*:
   Collection of specimens for complement fixation tests.

4. *Biochemistry*:
   (i) Qualitative Tests:
      (*a*) Examination of urine for protein, reducing sugar, acetone bodies, bile, blood *et cetera*.
      (*b*) Examination of gastric contents for presence of lactic and hydrochloric acid, starch, bile, blood and food residues.
      (*c*) Examination of fæces for occult blood.
      (*d*) Examination of blood for bilirubin (Van den Bergh reaction).
      (*e*) Examination of cerebro-spinal fluid for globulin.
   (ii) Quantitative Tests:
      (*a*) Blood: Determination of glucose, urea and creatinine content; determination of fragility of red blood cells.
      (*b*) Gastric contents: Titration of total and free acidity of fractional test meals.
      (*c*) Urine: Urea concentration and clearance tests for renal efficiency; tests for presence of reducing sugar in urine.
      (*d*) Cerebro-spinal fluid: determination of content of glucose, chlorides and protein.

Demonstrations.

Demonstrations may be given of the following:

1. *Hæmatology*:
   The use of the hæmatocrit.
2. *Bacteriology*:
   (*a*) Pneumococcal typing.
   (*b*) Cultural identification of bacteria.
3. *Serology*:
   (*a*) Complement fixation tests (Wassermann reaction *et cetera*).
   (*b*) Flocculation tests (Kahn test).
4. *Biochemistry*:
   (*a*) Qualitative tests: Lange's colloidal gold test.
   (*b*) Quantitative tests: Estimation of the diastatic index; estimation of calcium, phosphorus and cholesterol content of the blood; estimation of the basal metabolic rate.
5. *Miscellaneous*:
   Aschheim-Zondek test (Freidman modification), Schick test, Casoni test, cytology of the cerebro-spinal fluid, the von Pirquet reaction *et cetera*.

Conditions.

The course will be held at the Kanematsu Institute of Sydney Hospital, and will be limited to eight members only. It will not be held unless four applications are received.

The fee for the course is five guineas. Those wishing to attend should make application, enclosing cheque, to the Secretary, New South Wales Post-Graduate Committee in Medicine, the University of Sydney, before October 20, 1938.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on September 5, 1938.

Appointments.

The following appointments were approved: Dame Constance D'Arcy as the representative of the University of Sydney on the Council of the New South Wales Institute of Hospital Almoners; Dr. J. C. Eccles as an Honorary Lecturer in Physiology.

New Prizes and Scholarships.

The William Henry and Eliza Alice Sharp Prize.

The William Henry and Eliza Alice Sharp Prize, founded in 1938 by a bequest of £150 from the late Dr. Walter Alexander Ramsay Sharp, is to be awarded annually, on the recommendation of the examiners and of the Professor of Surgery, to the student who obtains the highest marks in clinical surgery at the final degree examination in the Faculty of Medicine. Value, £6.

The A. E. Sendall Bequest.

Under the will of the late A. E. Sendall, the sum of £1,000 was left to the University for the purpose of founding in perpetuity a scholarship or bursary in memory of the father of the late Alfred Edward Sendall, to be known as the "Horace John Sendall Scholarship or Bursary". The interest on the sum of £1,000 is to be used for such purposes as the Senate of the University may from time to time see fit. It has been decided: (i) That the interest on the capital be used for the provision of a bursary and not a scholarship, and that the bursary be known as the "Horace John Sendall Bursary". (ii) That the Horace John Sendall Bursary be tenable in one of the Faculties of Law, Medicine, Engineering, Dentistry, Veterinary Science or Architecture. (iii) That the annual value of the bursary be £40. (iv) That the conditions on which the bursary is conferred be those which apply to university bursaries generally.

Degree of Master of Surgery.

The Senate has approved recommendations by the Professorial Board and the Faculty of Medicine that the degree of Master of Surgery (M.S.) be conferred upon Dr. Stanley Hains Lovell, M.B., B.S.

The examiners were Sir John McKelvey, Dr. H. R. G. Poate, Dr. G. Bell, the Dean of the Faculty of Medicine (Professor H. R. Dew), and Professor W. K. Inglis. They reported that Dr. Lovell had fulfilled all the conditions prescribed for the degree.

MEETING OF CONVOCATION, THE UNIVERSITY OF SYDNEY.

A MEETING of the Convocation of the University of Sydney will be held in the Great Hall of the University on Friday, October 7, 1938, at 8 p.m. The meeting has been called to discuss the following matters:

1. Such report as may be presented by the senate of the university and/or the committee appointed by convocation to carry into effect the resolutions passed by the meeting of convocation held on June 10, 1936, upon all or any of the matters arising out of or relating to the resolutions passed by convocation on June 10, 1936.
2. The adoption of a constitution for convocation and the standing committee of convocation.
3. Ways and means of expediting the election of the first standing committee of convocation.

The committee appointed by convocation on June 10, 1936, to draft a suitable constitution for convocation and its standing committee, requests all graduates of the university of Sydney to be present at the above-mentioned meeting. Many matters of importance to graduates as a whole will be discussed.

The committee appointed by convocation comprised the following: R. Clive Teece, K.C., M.A., LL.B. (Chairman), A. M. Davidson, M.B., Ch.M., R. A. M. Allen, M.B., Ch.M., I. Clunies Ross, D.V.Sc., P. G. Spender, K.C., B.A., LL.B., M.P., Kevin Ellis, LL.B., H. G. Carter, B.E., M.Inst.C.E., M.I.E.Aust., Mrs. Kenneth Street, B.A., and Miss Monica Flower, M.A.

NOTICE.

THE Stawell Oration for 1938 will be delivered by Professor W. A. Osborne at the Medical Society Hall, Melbourne, on Wednesday, October 5, 1938. The title of the address will be "Ears, Eyes and Finger-Tips". The trustees responsible for the Stawell Oration wish as many members of the British Medical Association as possible to be present. An invitation is extended to the wives of those who propose to attend.

Books Received.

CLINICAL EXAMINATION OF THE NERVOUS SYSTEM, by G. H. Monrad-Krohn, M.D., F.R.C.P.; Seventh Edition; 1938. London: H. K. Lewis and Company Limited. Crown 8vo, pp. 338, with 111 illustrations, including 58 plates. Price: 8s. 6d. net.

THE DOCTOR'S VIEW OF WAR, edited by H. Joules, M.D., M.R.C.P., with a foreword by J. A. Ryle, M.A., F.R.C.P.; 1938. London: George Allen and Unwin Limited. Crown 8vo, pp. 122. Price: 3s. 6d. net.

DREAMS AND DREAMING, by H. T. Lovell; 1938. Sydney: Dymock's Book Arcade Limited. Crown 8vo, pp. 128. Price: 3s. 6d. net.

Diary for the Month.

Oct. 4.—New South Wales Branch, B.M.A.: Council (Quarterly).
Oct. 5.—Western Australian Branch, B.M.A.: Council.
Oct. 5.—Victorian Branch, B.M.A.: Branch.
Oct. 6.—South Australian Branch, B.M.A.: Council.
Oct. 7.—Queensland Branch, B.M.A.: Branch.
Oct. 11.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Oct. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Oct. 14.—Queensland Branch, B.M.A.: Council.
Oct. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
Oct. 19.—Western Australian Branch, B.M.A.: Branch.
Oct. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
Oct. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Oct. 25.—Queensland Branch, B.M.A.: Council.
Oct. 26.—Victorian Branch, B.M.A.: Council.
Oct. 27.—South Australian Branch, B.M.A.: Branch.
Oct. 27.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments.

Dr. C. G. McDonald has been appointed a Director of the Prince Henry Hospital, Little Bay, New South Wales.

* * *

Dr. R. I. Campbell has been appointed Honorary Urologist to the Liverpool State Hospital, New South Wales.

* * *

Dr. L. W. Wing has been appointed Government Medical Officer at Cooma, New South Wales.

* * *

Dr. N. A. Walker has been appointed Government Medical Officer at Gundagai, New South Wales.

* * *

Dr. K. W. Hodby has been appointed Medical Officer of Health to the Dowerin Road Board, in accordance with the provisions of *The Health Act*, 1911-1937, of Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xxii to xxiv.

Children's Hospital (Incorporated), Perth, Western Australia: Junior Resident Medical Officers.
Fremantle Hospital, Fremantle, Western Australia: Junior Resident Medical Officer.
Mater Misericordiæ Public Hospitals, Brisbane, Queensland: Honorary Officers.
Royal Hobart Hospital, Hobart, Tasmania: Resident Medical Officer.
Saint Joseph's Hospital, Auburn, New South Wales: Honorary Urologist.
The Broken Hill and District Hospital, Broken Hill, New South Wales: Resident Medical Officer.
Victorian Eye and Ear Hospital, Melbourne, Victoria: Staff Vacancies.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
South Australian: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to the Editor, The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, The Medical Journal of Australia, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognise any claim arising out of non-receipt of journals unless such a notification is received within one month.

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.